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### CARDIAC EMERGENCIES\*

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By definition, an emergency is "an unforeseen occurrence or combination of circumstances which calls for immediate action or remedy." For most patients with heart disease, such situations are rare. Quite commonly the patient's condition is such that remedial measures must be instituted if improvement is to be expected; but usually such measures, however appropriate, need not necessarily be immediate. Situations requiring immediate attention and thoughtful consideration arise much more frequently than those demanding immediate "action or remedy." Correct management may deny precipitate action—or new remedy of any sort. On occasion, indeed, the administration of some potent drug in a fancied emergency may create a situation much more critical than the one it was employed to correct.

While by strict definition cardiac emergencies are relatively rare, in the aggregate many situations arise in which prompt remedial action is imperative or in which the initiation of appropriate new or special measures should not long be delayed. Without too rigid adherence to the definition, certain cardiac emergencies and situations urgently calling for correct decision will be discussed briefly.

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## SHOCK

In most instances of acute circulatory failure the fault lies primarily in the peripheral circulatory apparatus, and not in the heart. Shock, therefore, as a rule, cannot properly be listed as a cardiac emergency, and its appropriate treatment is not a concern of this discussion.

Occasionally, however, the heart is affected by the toxic or other processes responsible for shock; in these instances it becomes a factor in the symptoms, and must be considered in therapy. In such cases, as in other instances of acute toxemia, chief reliance must be placed in measures generally applicable to the basic situation rather than in drugs which act specifically upon the heart. Vigorous therapy directed primarily to the heart is more likely to do harm than good. Morphine is perhaps more valuable, so far as purely cardiac effects are concerned, than caffeine.

In other instances of shock, as, for example, in certain cases of acute coronary insufficiency, insult to the heart is the primary factor. Treatment in these circumstances will be discussed in the following paragraphs.

## CARDIAC ARREST

**AURICULAR ARREST.**—With sudden shock or with strong reflex effects upon the vital centers, impulse formation at the pacemaker on occasion may be suspended for variable intervals. Unless spontaneous beating of the auricle or of the ventricle soon begins, serious symptoms ensue. In operative cases in which a more or less direct approach to the heart may be possible, *cardiac massage* may be restorative.

In some patients, stimulation of a *hypersensitive carotid sinus* may cause auricular arrest. Such stimulation apparently may occur spontaneously, from pressure of a small tumor, or with a certain turn of the head. Dizziness or fainting may result. In testing the sensitivity of the carotid sinus, unduly forceful digital pressure may cause cardiac standstill of alarming extent. Such testing, therefore, should be approached with caution.

*Atropine* in moderate dosage may be of great value in preventing the attacks. So may *ephedrine*, 0.02 to 0.05 gm.

three times a day or oftener. The removal of precipitating causes obviously should be attempted.

**VENTRICULAR ARREST.**—Temporary cessation of ventricular activity may occur in patients with severe heart disease, more commonly in cases with various degrees of *auriculo-ventricular block*. Ventricular asystole may be observed also as a part of the *carotid sinus syndrome*. Whatever the cause, absence of ventricular contraction for more than a few seconds produces alarming symptoms.

*Adams-Stokes seizures* may call for emergency measures during an attack as well as for efforts to prevent recurrence. Care applicable to convulsive seizures generally should be given to the patient. In an individual seizure the general prospect favors resumption of ventricular beating, and special measures directed toward that objective seldom are indicated. If, however, spontaneous beating appears improbable the injection of a few minims of *epinephrine* into the ventricular muscle should be tried. Such procedure on occasion has been followed by resumption of ventricular activity, but since spontaneous contractions may begin after they have been despaired of, it is obvious that the value of intraventricular injection of epinephrine can scarcely be regarded as established.

*Prevention of Recurrence.*—In every case, effort should be made to prevent recurrence of the attacks. Therapeutic measures directed toward prevention in the rarer instances associated with the carotid sinus syndrome have already been mentioned. In other cases *ephedrine*, 0.02–0.05 gm. three times daily or more frequently, or *epinephrine*, 0.3–0.5 cc. of 1:1000 solution, offers the best prospect. Occasionally *atropine* is efficacious. In a case associated with congestive heart failure the attacks ceased under *digitalis*. *Barium chloride* has been reported to be of value.

#### TRAUMA OF THE HEART

*Penetrating wounds* of the chest may produce a cardiac emergency by involvement of one of the heart chambers or by injury to the muscle. In either event, the production of *tamponade* from hemorrhage into the pericardial sac may

give rise to an increasingly grave situation, which in most cases that are not immediately fatal constitutes the most acute indication for immediate treatment. Characterized by a falling arterial pressure, a rising venous pressure, and in uncomplicated cases a "small, quiet heart," acute compression of the heart demands prompt relief by evacuation of the accumulated intrapericardial blood. The bleeding area must receive appropriate surgical treatment.

*Contusion of the heart* may result from nonpenetrating injuries to the chest. Common evidences of contusion are pain, dyspnea and change in heart rate, more commonly tachycardia. The heart sounds may be faint and may be altered in quality. Irregularity may develop. There may be abnormalities of any of the various components of the ventricular complex of the electrocardiogram.

In a suspected case the patient should be at rest, with administration of morphine if necessary, while studies are being made to determine the nature and extent of possible cardiac injury. Intrapericardial bleeding must be kept in mind.

#### NONTRAUMATIC TAMPONADE

In nontraumatic cases cardiac compression of any sort usually develops more slowly, but occasionally fluid accumulates in the pericardial cavity so rapidly as to require prompt removal.

**PURULENT PERICARDITIS.**—In many instances of nonpurulent pericardial effusion, there is no occasion for removal of fluid, and decision as to the best method of procedure usually may wait; but pus in the pericardial sac calls for evacuation. In questionable cases exploratory puncture should be made, any risk in such diagnostic process being less than that in delay when pus is present. Once the diagnosis is established, surgical procedures should not long be delayed.

#### DISSECTING ANEURYSM OF AORTA

Although under strict classification most instances of dissecting aneurysm of the aorta would be excluded from a discussion of cardiac emergencies, the subject yet may well receive brief consideration here. Recognized instances of dis-

secting aneurysm of the aorta with recovery probably will increase. Such possibility should be kept in mind particularly in cases of severe chest pain that are not readily explained; cases suggesting myocardial infarction but not conforming to the usual pattern; and cases in which the pain may be low in the back of the thorax or in the abdomen.

Pressure of the accumulated blood may interfere with the nerve or arterial supply below the site of the lesion. The electrocardiogram and the x-ray may be of great value in differential diagnosis, particularly by comparison of progressive records. At present the urgent indication is *utmost quiet*.

#### SYNDROMES RESULTING FROM DEFICIENCY IN CORONARY CIRCULATION

It is well recognized that a considerable restriction in coronary circulation often may be asymptomatic. When the blood flow is inadequate for myocardial function, symptoms develop. The disproportion between the need for and the supply of blood varies in duration and in degree, in cause and in effect. The clinical results likewise vary greatly. Cases in which the symptoms are of brief duration and in which they are precipitated under more or less well defined circumstances belong to the group commonly called "angina of effort." The predominant manifestation is an unpleasant sensation which may be agonizing or which the patient may refuse to call pain. In other instances, the ischemia resulting from deficient blood supply may be relatively more severe and of longer duration. There may or may not be occlusion of the arterial lumen from narrowing or from thrombosis without acute infarction. The symptoms may be more prolonged, or even continuous for a considerable time. Blungart has designated such cases of coronary insufficiency as "coronary failure."

In some instances the deficiency, with or without occlusion, may be so great as to produce acute infarction in the myocardium, resulting in characteristic signs and symptoms such as fever, leukocytosis, increased sedimentation rate, prolonged pain and electrocardiographic changes.

ANGINA OF EFFORT.—In an individual attack the probability of untoward event is small, and yet serious danger is inherent in ischemia of the myocardium. It appears to promote ventricular fibrillation or standstill. Any danger, however slight, should be obviated. Whatever the precise mechanism of the pain, therefore, it should be regarded by the patient as a signal for *cessation of the precipitating factor*, so far as this is possible. If walking, he should stop or slow his pace. *Nitroglycerin*, grain 1/200 or less, dissolved in the mouth, usually will cut short an attack and generally should be employed.

Better still, the attack should be prevented. In most cases careful analysis will indicate certain practices which the patient can change with a resultant diminution in the number of seizures. Taking nitroglycerin at the earliest intimation of onset, or even under circumstances which regularly induce an attack, is good therapy.

INFARCTION.—Although the immediate prospect in the average case of acute myocardial infarction is that the patient will improve, yet because of the serious possibilities, every case in its early phases must be regarded as an emergency. The work of the heart should be lowered as much as possible. *Protective measures* must be instituted. In certain instances precautions may be lessened in some degree after a time, but at first the patient invariably should be put *at rest* no matter how mild may be the evidences of infarction. Anything that increases coronary requirements or that diminishes coronary flow must be avoided. The possibility of reflex (vagus) interference with the coronary circulation should be kept in mind.

Abdominal distention is to be guarded against. Little food should be allowed at first. In most cases *morphine* is advisable. The dose should not be too small. There is considerable evidence to indicate that vagal effects tend to diminish coronary flow and thus even to extend the area of infarction—and that these vagal influences are lessened by *atropine*. Atropine administration would appear to be good therapy.

Because of the experimental evidence that phenobarbital tends to lessen cardiac output, *nembutal* probably is to be preferred in cases in which barbiturates are indicated. Unless

signs of congestive heart failure appear, digitalis should not be ordered. By most authorities the routine use of quinidine is not approved.

**PROTRACTED CORONARY INSUFFICIENCY (Coronary Failure).**  
—In those cases of frequent and severe pain in which it is apparent that, whatever the details, the deficiency of the coronary circulation is relatively very great, the patient should be fully protected and, for a time at least, should be at rest. In general, the treatment is that applicable to acute myocardial infarction. In some such cases infarction is impending. If no evidences of infarction ensue, and if satisfactory improvement follows, precautions accordingly may be lessened or suspended.

#### PAROXYSMAL TACHYCARDIA

In most cases there is no emergency. The great majority of attacks subside spontaneously. Simple sedatives or morphine along with reassurance of the patient usually meet all requirements.

**AURICULAR PAROXYSMAL TACHYCARDIA.**—In paroxysms of auricular origin, *carotid sinus pressure* frequently is effective. The initial application of pressure should not be too strong or too long continued. *Emesis* often stops the paroxysm. More rarely *quinidine* may be employed. In patients with little evidence of heart disease, if the attacks are of long duration, and if no undue susceptibility is shown after a small test dose, 0.2 gm. (grains 3) every three or four hours, or larger dosage, may be used.

In persistent attacks *acetyl-beta-methylcholine*, 10 to 30 mg., has sometimes been employed. It must be remembered that the drug is a powerful vagus stimulant and that serious symptoms may follow its use. In asthmatic patients it should not be employed under any circumstances. Atropine, grain 1/50 hypodermically, is the antidote.

In some cases digitalis in large doses has caused cessation of the attack.

**VENTRICULAR PAROXYSMAL TACHYCARDIA.**—Tachycardias of ventricular origin are rarely encountered except in cases of serious heart disease. In ventricular tachycardia of long dura-

tion the question of the employment of *quinidine* frequently arises. In considering the problem it must be kept in mind that it is by its depressant action on the muscle that the drug effects cessation of such abnormal rhythms. In some instances the risk attendant on this action under such circumstances may appear to be less than the probability of spontaneous cessation of the attack.

A large proportion of the reported cases of ventricular tachycardia have followed *digitalis intoxication*. In an individual instance, unless one can be sure that no *digitalis* has recently been administered, the giving of the drug obviously would be fraught with great danger. In other cases no good result is to be expected, and on theoretical grounds *digitalis* is contraindicated. If congestive heart failure supervenes, it might well be tried.

#### CONGESTIVE HEART FAILURE

There are no sharply defined criteria by which cases of congestive heart failures may be grouped according to severity. In a small proportion of cases, it is apparent at the first examination that immediate treatment is urgent. In most such instances *morphine* should be the therapeutic agent first employed. The administration of *digitalis* should await a comprehensive effort to obtain information regarding recent dosage. Under no circumstances should large doses be ordered except with the certainty that they may not be contraindicated by amounts previously taken.

In case of uncertainty as to recent dosage, *digitalis* should be given with great caution. If none has recently been given, it may be that maximum aid will require a considerable quantity; but some benefit will accrue from a lesser amount. Total dosage then may be reached more gradually.

If the patient cannot take or retain *digitalis* by mouth, careful administration *intravenously* may well be employed. But rarely indeed is emergency the indication for intravenous dosage. More than once unwise administration of intravenous *digitalis* preparations has produced emergencies of very grave import.

However much its accomplishment might be desirable, im-

mediate complete digitalization is never an emergency procedure.

**ACUTE LEFT VENTRICULAR FAILURE.**—Acute failure of the left ventricle may be evidenced by paroxysmal dyspnea, particularly nocturnal, in many instances accompanied by pulmonary edema. There may be associated bronchospasm. The prompt hypodermic administration of *morphine* usually stops the attack. Especially in those cases with bronchospasm the simultaneous use of *atropine* would appear to be of value.

Upon detailed examination common findings are hypertension, an abnormal electrocardiogram, alternation, gallop rhythm, rales at the lung bases, or other evidences of heart strain or of heart disease. *Digitalis* is indicated in order to improve myocardial efficiency and thus prevent recurrence of the attacks. The repeated intravenous employment of *mercurial diuretics* similarly is valuable.



## TREATMENT OF VASCULAR EMERGENCIES\*

WARREN P. ELMER, M.D.†

and

LEE B. HARRISON, M.D.‡

VASCULAR emergencies in cases other than those in which direct trauma is present usually are due to spasm, hemorrhage, embolism, or thrombosis, individually or in combination. The indirect causes or exciting factors are numerous and include atheromatous degeneration of the vessel wall, infectious vascular disease, hypertension, and vascular congestion as in congestive heart failure. Physical factors as in thrombosis of peripheral arterioles in frost-bite, and hematologic factors as seen in the hemorrhage of hemophilia, purpura hemorrhagica and related diseases, also contribute.

Cerebral hemorrhage, cerebral thrombosis and cerebral embolism are the most common cerebral vascular emergencies and often produce identical findings following their occurrence. At onset, however, they usually present different symptoms.

### CEREBRAL HEMORRHAGE

Cerebral hemorrhage may be extradural, subdural, subarachnoid, or intracerebral. The first three types are usually the result of trauma. The commonest cause of intracerebral hemorrhage is the rupture of a diseased vessel, usually with associated hypertension. The onset is always sudden and may take place during sleep, excitement, or exertion. The patient loses consciousness immediately in all but the mildest

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the early stages. *Adequate fluid intake* should be maintained by hypodermoclysis and venoclysis, 1500 cc. of normal saline under the skin and 1000 cc. of 5 per cent glucose intravenously being the average daily requirement. *Adequate elimination* through bowel and kidney must be maintained. *Rest* should be obtained by the use of magnesium sulfate, 2 to 4 cc. of 25 per cent solution, intramuscularly at intervals of two hours, or by careful administration of morphine or barbiturates. Bromides are difficult to administer as the large dosages required can be given only by mouth or tube. Chloral hydrate, 25 to 30 grains in 8 ounces of milk per rectum, may be efficacious.

*Restraints* are frequently required in order to keep the patient from thrashing around. An *ice cap* to the head can do no harm and may diminish the cerebral blood flow. *Tube feeding* may be necessary if coma persists more than twenty-four hours. *Venesection* early, especially in cases of hypertension, may be of value; 500 cc. of blood should be withdrawn slowly. Venesection should not be done in embolism or thrombosis. Frequent turning of bed patients, especially paralytics who are apt to be incontinent, is necessary to prevent decubitus ulcers. A retention catheter is frequently necessary.

### CEREBRAL THROMBOSIS

The main causes of cerebral thrombosis are as follows: (1) diseases of the blood vessels, the most common of which is arteriosclerosis, (2) abnormalities of the blood causing increased coagulability, (3) reduction in rate of blood flow and (4) trauma to the vessels themselves.

*Symptoms* usually develop gradually. Prodromal symptoms frequently occur in the form of dizziness, transitory attacks of aphasia or mental confusion. Following the onset of symptoms which may begin with numbness and weakness of an arm or side of the face, they may continue to increase for twenty-four to forty-eight hours before reaching their height. Loss of consciousness may occur then or later in the picture.

*Diagnosis.*—Hypertension may or may not be present. Syphilis is a frequent factor. The physical findings, when the thrombosis is located in the region of the internal capsule, are similar to those of hemorrhage.

cases. The physical signs depend on the site of the hemorrhage and its size.

The three main *types* of hemorrhage are (1) hemorrhage into the region of the internal capsule, (2) pontine hemorrhage and (3) hemorrhage into the cerebral ventricles.

*Hemorrhage into the region of the internal capsule* usually causes unconsciousness, the depth of coma and degree of shock depending upon the size and persistence of the hemorrhage. The pulse tends to be slow and respirations slow and stertorous. The head usually rotates and the eyes deviate to the side of the lesion. Flattening of the nasolabial fold usually can be noticed on the paralyzed side. The arm and leg become paralyzed on the opposite side from the lesion and are flaccid immediately following the attack. Positive toe signs appear on the paralyzed side. Incontinence of urine and feces is the rule.

*Pontine hemorrhage* produces facial paralysis on the side of the lesion and flaccid paralysis of the limbs on the opposite side. The patient with hemorrhage at this site lies with head and eyes turned to the side of the paralyzed limbs. Frequently the hemorrhage involves both sides of the pons and flaccid paralysis of the limbs on both sides with bilateral toe signs is present. Marked contraction of the pupils is usually seen.

*Hemorrhage into the ventricles* is not uncommon, and is due to leakage from hemorrhage in the region of the internal capsule. This type of lesion produces deep coma, and signs of pyramidal tract lesion are present on both sides of the body. Frequently it is quite difficult to differentiate it from pontine hemorrhage. The spinal fluid is bloody and under increased pressure. Glycosuria is not uncommon as a result of the cerebral lesion.

The *diagnosis* of cerebral hemorrhage in the acute stage involves the various causes of coma; it therefore must be differentiated from (1) cerebral embolism and thrombosis which will be discussed later, (2) diabetic coma, (3) uremic coma, (4) intracranial tumor, (5) postepileptic coma, (6) acute alcoholic coma, (7) drug poisoning, (8) hysteria, (9) skull injury and (10) encephalitis and meningitis.

TREATMENT.—Treatment is entirely supportive, at least in

known to substantiate such a diagnosis in certain cases. Acute hemiplegias, monoplegias and other phenomena are frequently seen in the absence of demonstrable cerebral disease. The rapid transient effect of such phenomena also makes it highly probable that they are caused by temporary spasm of the vessel rather than a pathologic condition like embolism or thrombosis. Again, certain cerebral symptoms appear to respond to vasodilators such as amyl nitrite and nitroglycerin.

Attacks attributable to spasm of cerebral arteries, especially the arteries supplying the region of the internal capsule, are, in our opinion, more common than is generally considered.

The *symptoms* or physical signs do not always present a definite picture and may vary from moderate headache, mild numbness of face, arm, or leg, or mild visual defects to complete hemiplegia. Consciousness may or may not be disturbed. Incontinence may occur, in addition to aphasia and other apoplectic phenomena. The patient usually recovers rapidly, often while awaiting medical aid. Recurrent attacks are common, though isolated attacks are frequently seen. Nervous states and anxiety appear to have some bearing on the condition.

**TREATMENT.**—Attacks are usually transitory and frequently have subsided before medical aid can be obtained. The cases which persist for a time appear to be benefited by inhalation of *amyl nitrite*, or by *nitroglycerin*, grain 1/100, hypodermically. *Rest* is important and mild *sedatives* such as phenobarbital, grain  $\frac{1}{2}$ , three times daily, or sodium bromide, grains 15 three times daily, should be given. The patient usually is markedly worried and anxious over the succession of events which have just taken place. Proper psychic handling to obtain assurance is worth the physician's time and patience.

#### INTRACRANIAL ANEURYSM

Rupture of an intracranial aneurysm usually proves rapidly fatal. Occasionally the patient survives to make complete recovery or to succumb later from subsequent leakage. The intensity of the symptoms varies according to rapidity and persistence of hemorrhage depending on the intracranial pressure. Loss of consciousness occurs when bleeding is considerable,

**TREATMENT.**—Treatment may be more efficacious than in hemorrhage because the patient usually is not in coma and can be handled more efficiently; otherwise, there is little difference in the treatment and handling of thrombosis and hemorrhage.

#### CEREBRAL EMBOLISM

Embolism of a cerebral artery may be due to all of the factors influencing the production of emboli. Blood clots within the circulation, such as those contained in an aneurysm, an atheromatous ulcer of a blood vessel, thrombi in a fibrillating auricle, vegetation of ulcerative endocarditis, thrombosis of a pulmonary vein and fat emboli following fractures, are some of the sources of emboli.

The *left middle cerebral artery* is the most common site of cerebral emboli. The point at which the embolus is arrested would depend on its size. A large clot may be arrested in the internal carotid or a small one may pass to a cortical branch. If the embolus is infected, cerebral abscess or meningitis may ensue.

**Symptoms.**—The onset is sudden, more so even than in cerebral hemorrhage but loss of consciousness is not so common. A convulsion may occur and headache is usually present. Hemiplegic signs appear if the capsular artery is affected. Prognosis is usually not so grave as in hemorrhage and the patient rarely dies. Complications like development of abscess or meningitis from infective emboli are of the most concern.

**TREATMENT.**—*Rest and general measures* as outlined for the treatment of cerebral hemorrhage also apply to this condition. If infection, such as thrombophlebitis or endocarditis, is present, measures should be instigated at once to combat it, since the brain is a fertile field. The *sulfonamide* group of drugs may be of distinct value here. Frequent *lumbar punctures* may be required for drainage in cases of meningeal infection. *Operative* interference may have to be resorted to in event of brain abscess.

#### CEREBRAL ARTERIAL SPASM

While spasm of the cerebral vessels in the human has not been proved conclusively, nevertheless, there are enough facts

from some point in the peripheral venous circulation, commonly from a thrombosed vein as seen in puerperal sepsis, pelvic inflammatory disease and thrombophlebitis. If the embolus is large enough, one of the pulmonary arteries may be completely obstructed, causing marked pulmonary edema which is usually fatal. Smaller emboli block smaller vessels of the pulmonary arterial tree and cause hemorrhagic infarcts of various size and severity. Cases of severe trauma, operative surgery particularly in the pelvis or involving the stomach, and bacterial endocarditis are conditions most likely to produce embolism of the lung.

*Pulmonary thrombosis* may appear when the lung is already the seat of chronic passive congestion such as that seen in chronic heart disease with congestive failure. The patient suddenly notices pain or tightness in the chest, commonly sub-sternal; cyanosis and dyspnea develop rapidly in severe cases. Cough develops with the production of bloody sputum followed by fever and leukocytosis. Friction rub is usually present. Death may occur quickly or protracted illness with recovery may take place.

**TREATMENT.**—Treatment is directed to *quieting the patient* as soon as possible. Morphine is usually the drug of choice in spite of reports of collapse in the severe cases. Immobilization of the chest with an *adhesive splint* frequently gives relief. *Thoracentesis* should be done only in the event of respiratory embarrassment from pleural effusion. Inhalation of *oxygen* may be of great benefit. If elevation of temperature continues and secondary pneumonia occurs, *sulfathiazole* or *sulfapyridine* may be given. In our experience an initial dose of 30 grains and thereafter 15 grains four to five times every twenty-four hours has maximum value in average cases. In hospital practice, determination of the sulfonamide blood level is of value in keeping the dosage at optimum.

#### RUPTURE OF AORTA

Spontaneous rupture of the aorta may occur in syphilis, bacterial invasion, or atheromatous necrosis of the media. Hypertension when associated with these conditions constitutes an added strain. Trauma from strain or external violence may

while in bleeding from a small aneurysm, slowly increasing headache and development of focal signs may occur, depending on the vessel involved.

**TREATMENT.**—Treatment here depends on *absolute rest*, the administration of morphine being indicated to insure it and to control hemorrhage if possible. *Surgical interference* may be life-saving, but it usually cannot be carried out during the stage of hemorrhage.

#### THROMBOSIS OF THE INTRACRANIAL SINUSES

Thrombosis of the intracranial venous sinuses is usually due to infection which has spread from some neighboring structure; or it is the result of direct injury. The lateral sinus, the cavernous sinus and the superior longitudinal sinus may be involved. The lateral sinus is the most commonly affected of the three; and infection of this sinus is virtually always the result of extension of a mastoid infection.

**Symptoms.**—Headache and pain in the ear, accompanied by rapid rise in temperature, are the first symptoms. Vomiting may occur. Congestion is noticed in the region of the mastoid and extension of the phlebitis to the jugular vein causes tenderness in the neck. *Focal symptoms are not pronounced as a rule.* The patient becomes toxic and occasionally delirium is present. Extradural or subdural abscess may occur. The prognosis is grave; however, it is better than in cavernous or longitudinal sinus thrombosis, recovery from the latter two being rare.

**TREATMENT.**—Treatment is directed first to the source of infection, namely the mastoid. *Ligation of the jugular vein* is usually done as a safeguard against pyemia. *Surgery* must be resorted to and the region of the sinus explored when dural abscess is suspected. *Chemotherapy*, that is, the use of sulfonamides and allied drugs, may be of value in combating the infection.

#### PULMONARY THROMBOSIS AND EMBOLISM

*Pulmonary embolism*, which is more common than thrombosis, is the result of the lodging of an embolus in the pulmonary arterial system. The embolus usually is a blood clot

may not be noticeable for days. Acute hemorrhage may be violent, with vomiting of large amounts of bright red blood or "coffee-ground" material. The patient may go rapidly into shock, with marked lowering of blood pressure, cool perspiring skin, weak thready pulse and obvious anemia. The size of the vessel affected and persistence of the hemorrhage determine the clinical picture.

*Diagnosis* is obvious in hematemesis: however, gross and occult blood in the stools may be indicative of bleeding anywhere in the gastro-intestinal tract above the rectum. In hemoptysis the blood is bright red, frequently frothy and in epistaxis examination of the nose and throat frequently reveals the source of the bleeding. Infected gums frequently cause considerable slow hemorrhage which may be swallowed and later vomited. X-ray examination of the gastro-intestinal tract may reveal the underlying disease if and when such procedure is possible. Proctoscopic examination should be made in suspected lower bowel bleeding. Gastroscoy if available is of value in some instances.

**TREATMENT OF ACUTE GASTRIC HEMORRHAGE.**—In acute cases immediate treatment is imperative. *Absolute rest* must be obtained at once. Morphine, grain  $\frac{1}{4}$ , should be given hypodermically and repeated as necessary.

*Replacement of blood volume* should begin at once, transfusion of whole blood being the method of choice. Blood plasma is also recommended, and it has the advantage of not requiring a typing routine and thus is more quickly available. Acacia, 100 cc. of 30 per cent solution, has been given intravenously to replace blood volume and maintain blood pressure; however, in our experience it has been of no more value than intravenous glucose, 500 cc. of a 5 to 10 per cent solution of the latter being injected every eight to twelve hours.

There are numerous *hematinics*, such as thromboplastin, snake venom, various calcium compounds and so-called blood coagulants, which have been reported as being more or less effective in the control of hemorrhage. They usually prove to be of little value in gastric bleeding. *Stimulants* such as adrenalin should be used with extreme care, if at all, in the shock following mesenteric hemorrhage. Caffeine sodium

be the final precipitating factor in the rupture of a diseased aorta. In twenty-four cases of rupture of the aorta recorded by De Vries, there were ten cases of rupture of a syphilitic aneurysm, five of rupture of the aorta from esophageal carcinoma, four traumatic ruptures and five so-called spontaneous cases.

*Symptoms* are dramatic and rapid with sudden shock, rapid loss of consciousness, and death in cases of complete rupture.

*TREATMENT.*—Treatment is purely *preventive*, and consists in treatment of the aortic disease and restriction of activity when known disease is present.

#### GASTRIC AND ESOPHAGEAL HEMORRHAGE

Gastric hemorrhage is a form of vascular emergency in which rarely the bleeding vessel itself is the site of disease. For this reason hemorrhage from the stomach is more a symptom than an entity, though it constitutes one of our gravest vascular emergencies.

The *causes* of gastric and duodenal hemorrhage are numerous, the most important being as follows:

1. Gastric or pyloric ulcers.
2. Gastric and esophageal tumors, particularly carcinoma.
3. Acute gastritis associated with persistent vomiting.
4. Direct severe trauma such as a severe blow on the abdominal wall or wounds of the stomach.
5. Ingestion of poisons such as bichloride of mercury or foreign substance such as glass.
6. Hepatic diseases, particularly cirrhosis with varicosities of the gastric and esophageal veins.
7. Various infectious diseases such as malaria and yellow fever, even in pneumonia gastric hemorrhage is seen and measles, mumps and scarlet fever may produce bleeding from the stomach.
8. Blood dyscrasias like purpura hemorrhagica and the leukemias.

*Symptoms.*—Slow hemorrhage may be present for hours or for a day or more before it is suspected. The first evidence is a dark, tarry stool. Weakness which is slowly progressive occurs and absence of gastric pain or discomfort is common. Pallor

gress more slowly than in embolism; however, in the presence of infection, massive extension of the thrombosis may occur. Frequently gangrene of a large section of the intestine is produced before the condition is recognized.

*Diagnosis* is made on the abdominal symptoms and course in the presence of vegetative valvular disease, arterial disease, abdominal operations, abdominal and pelvic infections, and other causes of thrombosis and embolism.

*TREATMENT.*—Small vascular occlusions of the mesentery usually cause little harm inasmuch as adequate collateral circulation develops. Acute pain is controlled by *morphine*. *Warmth* should be applied to the abdomen and *general supportive care* given. Symptoms usually subside in a few days to two weeks. Large occlusions and multiple areas of thrombosis usually result in severe damage to or gangrene of the part of the intestine supplied by the occluded vessel. In such cases, *operation* is advisable as soon as one can be reasonably certain that the condition is present.

#### ARTERIOSCLEROSIS OBLITERANS

Arteriosclerosis obliterans is found more frequently in males over sixty, and the excessive use of tobacco is less commonly admitted than in thrombo-angiitis obliterans. The arteries show more thickening and are more easily demonstrated in roentgenograms of the extremities. Thrombosis is rarely encountered in arteriosclerosis obliterans but a differential diagnosis based upon signs and symptoms is often impossible. The treatment of the two conditions is virtually identical.

*TREATMENT.*—During the last five or six years treatment has emerged somewhat from the chaotic state which so often occurs when new methods are advocated and no treatment is specific. Certain methods in the hands of some observers still seem to produce better results than the rank and file are able to obtain but, on the whole, experience is eliminating the more unsound methods of treatment. Cases without ulceration or gangrene are more amenable to any treatment. These cases may not, strictly speaking, be considered emergencies but any or all treatments may be applicable to any stage of the disease.

*Elimination of Tobacco.*—No treatment of arteriosclerosis

benzoate may be given hypodermically, or a solution of warm coffee may be given by rectum. *Elevation of the feet* is of value, and *external heat* in the form of electric cradle, electric pad, or hot water bottle should be given though care must be taken to avoid burns. An *ice cap* to the upper abdomen is of theoretical benefit and should be used except in shock cases.

*Diet.*—The patient is allowed only cracked ice and a few sips of alkalized water or milk the first twenty-four hours, following which, if improvement continues and hemorrhage has stopped, feeding is begun and increased quite rapidly. On the second day 8 ounces of milk and cream four or five times daily may be given with an alkaline powder such as magnesium trisilicate, one teaspoon after each feeding. On the third day the patient may have soft eggs, toast and milk, jello, cup custard, and well cooked cream of wheat. Increase of diet will then depend on improvement of patient.

**TREATMENT OF CHRONIC GASTRIC HEMORRHAGE.**—In slow gastric hemorrhage treatment is much the same; that is, complete rest must be obtained. Morphine usually is not necessary and transfusion may be avoided. The diet may be increased even more rapidly than in the acute cases. In mild hemorrhage, with the patient on diet and rest, the blood count may be normal within a week or so. *Iron* (ferrous sulfite, grains 3, or iron with liver) should be given three times daily when anemia is present.

#### MESENTERIC THROMBOSIS AND EMBOLISM

Thrombosis of the vessels of the mesentery occurs following inflammation of the bowel, mesentery, peritoneum and surrounding structures. Trauma and abdominal surgical procedures also are frequent factors. Embolism of a mesenteric artery may be due to any of the sources of emboli previously discussed.

*Symptoms* are acute, more so in embolism than in thrombosis. Abdominal pain with rapid development of shock occurs if a large vessel is occluded. A rapid bounding pulse, cool perspiration, drop in blood pressure and later a rise in temperature usually are seen. In thrombosis, the symptoms may pro-

with the patient in the recumbent position. Arm or leg veins are equally satisfactory. A different vein should be used for each administration, whenever possible, to avoid thrombosis. A flow of 10 to 15 cc. per minute has been found sufficiently rapid. No discomfort beyond mild flushing and thirst is experienced. The quantity is increased by 50 cc. at each injection until a maximum of 300 cc. is reached. The concentration is then increased by 0.5 per cent at each injection until 3 or 3.5 per cent strength is reached. Injections should be given every other day for two weeks and then twice a week for about three months. We have noticed marked improvement in several patients when no other treatment except elimination of tobacco was used.

*Buerger's Exercises.*—Buerger's exercises consist of lying on the back with thighs and legs at right angles to the body for two minutes, dangling the legs over the side of the bed in the sitting posture for three minutes and rest in the recumbent posture for three minutes. This procedure is to be repeated three to five times at intervals of four to six hours. The discomfort and pain are often greatly relieved by these exercises and after them the feet often assume a normal color for an indefinite period. These exercises should be continued for months, even years.

*Sanders Oscillating Bed.*—The Sanders oscillating bed performs much of the same service, alternately elevating and lowering the head and feet at slow speed and without muscular effort on the part of the patient. It can be recommended for aged or weak patients, who can continue the treatment for several hours at a time without fatigue. The chief disadvantage is the initial expense which is often unnecessary for ambulatory patients.

*Pressure and Suction Apparatus.*—Intermittent negative and positive pressure by means of various types of pressure and suction apparatus has been enthusiastically recommended by some observers. We have been disappointed in the results obtained, few patients having been benefited and actual damage to vessels occasionally has occurred. In our opinion, it has no advantages over Buerger's exercises or Sanders bed.

*Physical Therapy.*—*External heat* by means of electric lights

obliterans should be undertaken without the elimination of tobacco in all forms. Why the majority of smokers, especially women, are free from manifestations of arteriosclerosis obliterans is unknown, but once an individual exhibits signs of the disease, they are doomed to forego tobacco if treatment is to be successful. A few years ago many observers scoffed at the idea that tobacco was an important factor but today few if any with extensive experience deny the fact.

*Diet.*—Excessive use of condiments and spices should be controlled, but on the whole, except in diabetes, diet plays very little part in the treatment. *Alcohol* in moderation, especially an ounce of whiskey with plain water once or twice a day, seems of definite value.

*Exercise.*—Exercise must be restricted sufficiently to avoid pain but bed rest is not necessary in most cases.

*Vasodilator Drugs.*—Vasodilator drugs are of little use but combinations such as  $\frac{1}{4}$  grain of phenobarbital with 3 grains of theobromine or  $\frac{1}{2}$  grain of amytal with 2 grains of theamin, two or three times a day, may be of some benefit to depressed or apprehensive patients. *Aspirin* in 5- or 10-grain doses is our most useful drug for the relief of pain, since the chronicity of the disease, in our opinion, contraindicates the use of opium in any form except under unusual circumstances in which case it should be administered by the physician. De-insulinized pancreas and other *tissue extracts* have proved of little value in our hands and have been eliminated entirely from our treatment.

*Typhoid Vaccine Therapy.*—Intravenous administration of typhoid vaccine, each dose containing 20,000,000 to 40,000,000 organisms, has been recommended but in our experience the method has not proved of sufficient value to counterbalance the possibility of an increased sclerosis following repeated fever shocks, especially in older patients.

*Intravenous Injections of Hypertonic Salt Solution.*—We have found useful the intravenous administration of hypertonic saline solution, except in instances of advanced nephritis and marked arterial hypertension. The treatment should begin with 100 cc. of 1.5 to 2 per cent sodium chloride in distilled water. It is best given from the ordinary salvarsan apparatus,

Buerger's disease presents itself in the rapidly extending thrombosis with gangrene or following sudden occlusion of a major artery. It is estimated that sudden arterial occlusion of a large artery occurs approximately in 5 per cent of patients with established Buerger's disease. *Amputation* is necessary in about 60 per cent of these patients whereas, in occlusion in young people without Buerger's disease, amputation is usually necessary in only about 10 per cent.

The immediate treatment is directed toward protection of the extremities, re-establishment of the circulation, alleviation of pain and relief of secondary spasm. In gangrenous and pre-gangrenous states, conservatism is advocated by most clinics. Collateral circulation in young individuals often develops rapidly and with supportive care including heat, rest, careful attention to sloughing tissues, vasodilator drugs and hypertonic saline solution, amputation of a toe or leg can frequently be avoided. In our experience one of the most effective measures to keep the patient as symptom-free as possible is *Buerger's exercises*; and other methods as outlined under arteriosclerosis obliterans also apply in the treatment of this condition. Complete abstinence from tobacco is advised.

#### RAYNAUD'S DISEASE

This phenomenon is characterized by *redness and cyanosis of fingers and toes* which appear spasmodically, often associated with ulceration and focal gangrene. No organic disease or occlusion of the larger arteries is present. Cyanosis is produced by complete interruption of blood flow due to spasm of the digital, palmar, or plantar arteries. The etiology is unknown. The disease occurs more often in females, the rate being about ten to one. Patients who are underweight and under mental strain are more prone to develop it. The use of tobacco bears no relationship to its cause.

*Diagnosis* is made on the basis of cyanosis and pallor of the digits which come on in sudden attacks and are induced by cold and sudden nervous states. Symmetrical or bilateral involvement of digits, absence of occlusive arterial disease, gangrene limited to small areas of skin if present and greater incidence in females are other factors.

in cradles, electric pads or hot water bottles must be used with great caution since the extremities of these patients are more easily injured than those of normal persons. The temperature in a cradle should not exceed 105° F. at any time.

*Diathermy* in the hands of expert physiotherapists is of some value but it is dangerous when administered by those unskilled in its use. Gentle *massage* is often soothing but deep massage like suction may injure the vessels. The diabetic with arteriosclerosis obliterans should be more carefully observed but diabetic arteriosclerosis has no special form of treatment.

*Ulceration and Gangrene.*—When ulceration or gangrene is encountered the patient should be confined to bed. External applications to relieve pain are seldom efficacious but the oscillating bed and Buerger's exercises often give relief. The temptation to use opiates should be resisted by both the patient and the physician. Spontaneous amputation has not occurred so frequently in our experience as in that of Samuels'. Sixty per cent of our cases of well developed gangrene have come to amputation. When diabetes complicates the picture the prognosis is still more grave. Amputation, we feel, should be delayed as long as there is reasonable hope but, when performed, it should be above the knee.

#### THROMBOANGIITIS OBLITERANS (BUERGER'S DISEASE)

Buerger's disease is essentially an obliterating endarteritis and endophlebitis occurring chiefly in young males. The condition is chronic and intermittent and frequently presents acute attacks of phlebitis or cutaneous nodules. A progressive, migrating arteritis with secondary thrombosis and canalization is present. Acute forms frequently lead to gangrene with rapidly ascending thrombosis, whereas a chronic form may be very slowly progressive with exacerbations and remissions over a period of years. The etiology of the disease is unknown; however, clinical observations lead to the conclusion that a possible vascular sensitization may be present. Contributing factors may be numerous, among which may be listed tobacco, bacteria and ergot.

*TREATMENT.*—The necessity for emergency treatment in

**TREATMENT.**—The *forcing of fluids* helps to eliminate the drug and in mild cases this is all that is necessary. In severer forms of poisoning the treatment is the same as that of in thrombosis of arteries of the extremities.

#### PERIPHERAL THROMBOPHLEBITIS AND VENOUS THROMBOSIS

Thrombosis frequently occurs in the peripheral veins, usually as a result of stasis or infection. Dilatation of the veins often is the result of stasis from obstruction within the vein itself or from pressure without. Gravity also plays a part, particularly in the veins of the lower extremities, the most frequent site of varicose veins.

Thrombosis of a vein, particularly if infection is present, presents a hot, tender, swollen region at the site of the thrombosed area. General symptoms such as fever, and evidence of toxicity is present. There is leukocytosis. The chief cause for concern in these patients is the danger of pulmonary embolism.

Peripheral thrombophlebitis may occur over any venous area in sporadic form or it may accompany diseases like typhoid fever, or pelvic inflammations, or it may occur during pregnancy or puerperium.

**TREATMENT.**—Treatment consists of absolute rest of the parts involved, usually for a considerable period until the acute process has entirely subsided. If a limb is affected it should be elevated on a pillow. Cold applications are advisable and mild sedatives, usually in the form of barbiturates, should be given for rest.

**Varicosities.**—Varicosities, particularly of the lower legs, are favorable sites for thrombosis and occasionally thrombophlebitis, and they in turn lead to further venous dilatation. Additional causes of the condition are childbirth, pelvic tumors, obesity, tight garters, and work which demands considerable walking. Treatment consists of the following: (1) Injection of an irritating substance such as sodium salicylate, glucose, quinine and urethane, or sodium chloride in hypertonic solution. These irritants cause thrombosis and organization with shrinking and obliteration of the veins. This treat-

ment is now the method of choice. (2) Supportive bandages and stockings are of value before and after injection. (3) Operative removal of the dilated veins. This procedure was formerly the method of choice, but it has been largely replaced by injection therapy. (4) Rest and elevation of legs for temporary relief. (5) Weight reduction if patient is overweight. (6) Surgical removal of infected thrombus.

## RENAL EMERGENCIES\*

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In this discussion of renal emergencies, emphasis will be placed on the unlooked-for contingency which brings about a sudden demand for action. The long-recognized facts involving etiology and theories will be omitted. No effort will be made to make this an exhaustive and all-inclusive treatise but rather a clinic in which a few of the more recently recognized treatments are exemplified.

The kidneys and other excretory organs, namely the calices, pelves, ureters, bladder and urethra, are essential to life. Since excretion is the prime function of this system, interference with this function constitutes a major or minor emergency, according to the extent of the interference. Because it is essential, nature has provided an extravagant reserve, and all too frequently this reserve is being gradually and steadily diminished without manifesting this loss to the patient. It is estimated that there are two million glomeruli in each kidney and that about one fifth of these are in action at one time or are required for the continuation of life. The average rate of filtration of a glomerulus is 120 cc. per minute, and the average flow of urine is somewhat more than 1 cc. per minute. The difference is the reabsorption of fluid by the tubules. The average pressure of urine going from the kidney to the bladder is 50 mg. An emergency is created when there is major interference with this secretion or flow of urine.

### ACUTE RETENTION OF URINE

#### Acute Retention of Urine Due to Benign Hypertrophy of the Prostate Gland

CASE REPORT.—A patient, aged seventy-six years, was rushed to the hospital from a neighboring community. The man was in

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apparent agony, with cold perspiration over his face. His physician had made an unsuccessful effort to pass a catheter and relieve sudden complete urinary retention. Percussion revealed bladder dullness extending to umbilicus. A stiletted curved No. 22 Foley catheter was manipulated into the bladder in about two minutes, but still no urine escaped. It was evident that blood clots as well as urine had accumulated in the bladder and blocked the eye of the catheter. Using a large-bulb syringe, 2 ounces of boric acid solution were forced in with one sudden quick effort, whereupon the urine flowed freely. Care was taken to remove only 4 ounces, which gave immediate relief from the overdistention. The patient was placed in bed, and gradual decompression was accomplished by the simple method of removing 4 ounces every two hours and replacing 2 ounces of boric acid solution. In twenty-four hours continuous drainage was instituted. Rectal palpation revealed an enlarged prostate gland, smooth and soft, therefore, probably benign. Benign prostates bleed easily because of congestion whereas prostates with malignancy rarely cause bleeding since they are usually scirrhus and avascular.

*Comment.*—The experience of this patient briefly illustrates a few seldom-utilized facts in the treatment:

1. Effective entrance into the bladder is obtained by using a *curved* catheter. The size is unimportant. A semirigid catheter with a Coude tip will often suffice. A semirigid stilette inserted into a Foley catheter gives the necessary stiffness and a curve simulating the contour of the urethra produced by the bulging prostate. The stilette is removed and the bulb is distended, thus keeping the catheter in place without adhesive. Changing the curve of the stiletted catheter and carrying it well down between the legs and even elevating the end by a finger in the rectum is necessary in maneuvering the catheter by the elevated urethra.

2. Undue force is unwarranted since *extravasation* of urine may bring about another emergency which calls for immediate operation by multiple bold incisions into the extravasated area.

3. Suprapubic cystotomy or trocar suprapubic puncture by the inexperienced on the urgent plea of the distressed patient to do something, is not warranted.

4. Spontaneous rupture of the bladder does not occur.
5. Hemorrhage, shock and anuria may result from a sudden removal of all the urine.
6. The removal of a blood clot blocking the eye of the catheter is best accomplished by forcing fluid through the catheter and not by sucking it into the lumen of the catheter.
7. A bladder *filled* with clots can only be emptied of clots by a *metal* catheter of good caliber with which sufficient suction to remove the clots will not cause the collapse of the sides as occurs with a rubber catheter.

#### Acute Retention Due to Stricture

Stricture of the urethra causing acute retention is relieved by an entirely different procedure. This requires *small-caliber* catheters or filiforms, avoiding blind pockets, manipulating by corkscrew curves. Patience in trying out many filiforms is usually rewarded by one slipping by. My former associate, Bransford Lewis, cautioned, "When an advantage is gained, do not lose it!" The filiform strapped in for twenty-four hours will permit the trickling of sufficient urine around it to relieve the patient, and it is surprising how easily a fairly large sound can be passed twenty-four hours later due to the lysis of the fibrous strictured area. A La Forte follower may be used but care must be taken that the threads are not so worn as to permit the follower to slip away into the bladder.

#### Acute Retention Following Pelvic Operations

The acute retention following lower abdominal or rectal operations is the result of the temporary paralyzing of the parasympathetic to the detrusor muscles and pudendi nerves. The use of prostigmine, 25 mg. hypodermically, has been rather disappointing although theoretically it should be quite effective. The author prefers to catheterize the patient frequently without irrigations.

Recent experimental work performed at the St. Louis City Hospital by the resident urologist, Reese Coleman,<sup>1</sup> indicates that 2 gm. of the sodium salt of *sulfathiazole* given orally is absorbed in adequate amount in ten minutes, and 100 consecutive patients given 1 gm. of sodium sulfathiazole before

instrumentation had no reaction. Routine administration of 1 gm. of sodium sulfathiazole before catheterization or instrumentation is recommended. This will certainly prevent the postoperative cystitis so often seen.

*Trauma* at catheterization, due to carelessness or poor visualization of the urethra, causes more trouble than unsterile apparatus. The habit of instilling *silver salts* after catheterization is to be condemned, since decomposition of the silver causes irritation to the mucous membranes. A mild irrigating solution of *borosaline*—boric acid 3 per cent with normal saline is mildly antiseptic and isotonic—probably offers the best results.

### Acute Retention of Neurogenic Origin

The acute retention following spinal injury may be treated by one of three methods:

1. Permit the bladder to overdistend and thereby produce overflow incontinence and hope for the occurrence of urination by reflex action. Morphine and hyoscine must be given to relieve the pain of this overdistention before the overflow occurs, which usually takes three or four days.

2. Intermittent or continuous drainage by catheterization.

3. Suprapubic cystotomy.

The first-mentioned procedure is recommended in practically all the textbooks, to avoid introducing infection. The second measure, which usually results in the use of a permanent indwelling catheter, is very satisfactory in the female but not very practical in the male. The third measure, cystotomy, is the procedure of choice when any long-continued situation is expected. A cystotomy is not without shock in itself and should not be performed if superimposed on an already existing state of shock; instead, catheterization should be resorted to until the patient is in better condition.

With the recent use of drugs like mandelic acid and the sulfonamides, the danger of infection by catheterization or suprapubic cystotomy is greatly lessened. The persistent residual urine with its attendant back-pressure on the kidneys constitutes the real threat of resulting uremia and death. Cystotomy prevents this occurrence.

## RENAL COLIC

**CASE REPORT.**—A physician was brought to the hospital in an ambulance. Questioning revealed that while he was out on a call, a sudden severe, sharp-shooting pain in the right side, radiating to the groin, caused him to collapse, whereupon an ambulance called quickly brought him to the hospital.

The pulse was 98, temperature 100° F., respiration 24, blood pressure 198 systolic, 80 diastolic. A hurried examination revealed slight rigidity in the right lower quadrant but no exquisite tenderness over the appendix. There was slight tenderness in the costovertebral angle but none in the gallbladder area. The kidney could not be palpated. The lungs were apparently clear; the white cell count taken immediately showed 10,800 per cubic millimeter of blood. The differential count showed a mild infection: the testes, epididymes and inguinal rings were normal; the abdomen was distended. The urine voided upon arrival at the hospital was grossly clear; amber in color; reaction acid; specific gravity 1.020; no albumin or sugar; microscopic examination showed 10 red blood cells per high power field and a few pus cells.

The kidney, ureter and bladder roentgenogram (Fig. 46) showed a few small shadows on the opposite side of the pain. Pancreatic tissue extract (Depropanex) 3 cc., injected in the muscle gave relief in three minutes. Morphine given the patient before coming to the hospital only excited him. Catheters passed to both kidneys cystoscopically drained 4 cc. from the left and 12 cc. from the right, which was bloody. The shadows seen on the previous plates were found outside the ureteral catheter shadow. After injecting 8 cc. of 25 per cent argyrol, a faint shadow was seen in the x-ray film (Fig. 47); after withdrawing the catheter and injecting air, this faint shadow could be seen lying in line with the air-injected ureter (Fig. 48); a lateral x-ray confirmed this.

A uric acid stone (nonopaque to x-ray) was removed by the basket forceps which was cystoscopically manipulated up the right ureter.

**Comment.**—This case report is classical, but provokes these few remarks. Recent investigational work using the Trattner apparatus permitted us to establish the fact that morphine increased peristaltic waves in the ureter, and atropine and benzedrine neutralized them. *Pancreatic tissue extract* (Fig. 49) was found to relax the ureter in three minutes and clinically to re-

Fig. 46.



Fig. 47.



Fig. 48.

Fig. 46.—Roentgenogram showing no evidence of calculus in the urinary tract.

Fig. 47.—Following the injection of 25 per cent argyrol, the opacity is seen alongside the ureter and is indicated by the arrow.

Fig. 48.—The air pyelo-ureterogram taken after Fig. 47 shows the opacity (uric acid stone) within the ureter indicated by the arrow.

lieve the pain immediately. Pain in kidney colic is often the result of spasm at the site of the stone, and pancreatic tissue extract, acting apparently by neutralizing the cholinergic influence, relieves the spasm. This is a more desirable effect than that produced by morphine which takes sixteen minutes to act and then does so by cerebral numbing rather than by exerting its effect at the site of the lesion. *Avertin* (Fig. 50), 1 mg. dissolved in 50 cc. of sterile distilled water and injected through the catheter, will cause relaxation and dilatation of the ureter also. *Prostigmine*, 1 cc. of a 1:2000 solution in-

Fig. 49.

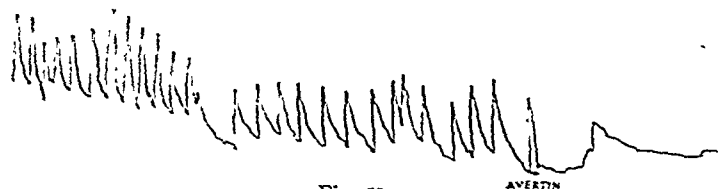
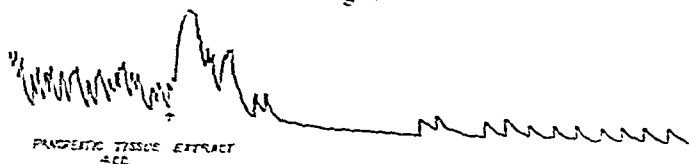


Fig. 50.

Fig. 49.—Kymographic tracing of contractions of the human intact ureter indicating complete absence of peristaltic waves three minutes following injection of 3 cc. of pancreatic tissue extract.

Fig. 50.—Kymogram showing complete absence of peristaltic waves in the ureter following the injection of avertin in the ureter.

jected intramuscularly, will increase contraction and has been known to expel small stones in this manner.<sup>2</sup>

The pain caused by renal colic becomes a real emergency in the eyes of the patient. The obstruction, if permitted to continue over forty-eight hours, becomes a real emergency to the physician since infection and loss of function may ensue. *Cystoscopic treatment*, with manipulation of a catheter beyond the stone to relieve the obstruction, is indicated after preliminary medication for relief of pain; if this is unsuccessful, *nephrotomy* or *lithotomy* should be performed with-

Fig. 46.



Fig. 47.



Fig. 48.

Fig. 46.—Roentgenogram showing no evidence of calculus in the urinary tract.

Fig. 47.—Following the injection of 25 per cent argyrol, the opacity is seen alongside the ureter and is indicated by the arrow.

Fig. 48.—The air pyelo-ureterogram taken after Fig. 47 shows the opacity (uric acid stone) within the ureter indicated by the arrow.

out delay. A stone in the ureter, no matter what the size, if found to be unmovable by manipulation, should be surgically removed at once because it is adherent to the wall of the ureter and all efforts at removal by manipulation, no matter how many, will be fruitless and harmful.

Although in the majority of cases renal colic is caused by a stone obstructing the outflow of urine in the ureter or pelvis, strictures of the ureter (Fig. 51), ureteral spasm, ptosis of the kidney with kinking of the ureter (Fig. 52) and external pressure, as by retroperitoneal tumor, may bring about the same symptoms. Acute hydronephrosis may follow the sagging of a kidney over an aberrant vessel, causing acute obstruction. This is demonstrated in Figure 53. Regurgitation of urine<sup>3</sup> occasioned by a sudden strain at unsuccessful voiding, with the urine forced back up the ureter, may cause typical renal colic. The relief for this condition is simply emptying of the bladder with a urethral catheter (Fig. 54).

A proper diagnosis must always follow the immediate relief afforded by the emergency and proper treatment rendered to prevent complete impairment or destruction of the organ.

### ANURIA

#### Recumbent Lithiasis

Complete anuria and death followed the development of bilateral stones in a 17-year-old boy following treatment of a fracture of a leg in which the patient remained in bed four months. No urine was passed when the patient began to walk on crutches because the stones, formed during his rest in bed, fell into the pelvic outlet and completely blocked the ureters. Recumbent lithiasis, well-known to the urologist, becomes an emergency in this situation and calls for bilateral nephrostomy immediately when recognized. A plain x-ray film will differentiate the condition from nephritis since these stones are always phosphatic. Ockerblad and Carlson<sup>4</sup> have recently reviewed such a condition.

#### Anuria Following Accidental Ligation of Both Ureters

This tragic accident occurs probably more frequently than reports in the literature would indicate. The ureter may be



Fig. 51.



Fig. 52.

Fig. 51.—Acute renal colic caused by hydronephrosis from stricture of the ureter—edema caused the sudden block.

Fig. 52.—Ureteropyelogram demonstrating an acute hydronephrosis due to a kink of the ureter. The patient narrowly escaped an appendectomy for this condition



Fig. 53.



Fig. 54.

Fig. 53.—Renal colic caused by sudden sagging of the kidney over an aberrant vessel.

Fig. 54.—Renal colic caused by regurgitation of urine following strain from unsuccessful attempts at voiding.

mented forms, 24 per cent stab forms, 17 per cent lymphocytes and 1 per cent mononuclears. No malarial parasites were found. A catheterized specimen of urine showed acid reaction, specific gravity 1.035, and no sugar, albumin, or red blood cells. The Kahn reaction was negative. Blood sugar was 87 mg. per 100 cc. and nonprotein nitrogen 18 mg. per 100 cc. Blood culture was negative. The sputum showed type VIII pneumococci.

A chest plate showed a pneumonic infiltration at the left base. On the day of admission the patient was given sulfapyridine, 15 grains (1 mg.) every hour for four doses, and 15 grains every four hours thereafter.

On the day following admission the temperature reached normal and remained so. On October 24 the red cells numbered 4,810,000 and the white cells 7300 per cubic millimeter of blood.

On October 26, after four days of sulfapyridine administration, the patient had gross hematuria and began to complain of abdominal cramps and pain in both lumbar areas. The abdomen was soft, with slight bilateral costovertebral tenderness. Sulfapyridine was discontinued. The output for the following twenty-four hours was only 100 cc. of bright red blood in spite of the administration of intravenous fluids. The patient vomited frequently.

On October 28 the urinary output was nil and the blood non-protein nitrogen was 53 mg. per 100 cc. Immediate cystoscopy was advised. This was performed under local anesthesia with the following findings: The bladder mucous membrane was congested throughout and several small, white, irregular, soft concretions were found on the floor of the bladder. These readily dissolved in warm water. A similar concretion was seen protruding from the left ureteral orifice and both orifices appeared lacerated. No. 6 French catheters were passed for 26 cm. on each side, and caused a gritty sensation during passage. No drip was obtained on the right side, and a slow, blood-tinged drip was obtained on the left.

A roentgenogram was taken which showed no opaque shadows. A pyelogram showed a complete filling defect in the pelvis of the right kidney with only a small amount of the dye apparent in the upper calix, the major portion of the dye appearing alongside the right catheter. On the left side no dye was seen in the renal pelvis or calices, but some appeared around the catheter, as on the right side. The No. 6 French catheters were withdrawn and No. 8's passed on both sides; the renal pelves were thoroughly lavaged with warm distilled water. Following

completely severed, ligated by a suture or injured by forceps, usually in the region of the broad ligament or at the insertion of the ureter into the bladder. Anuria following pelvic operations should arouse a suspicion of its occurrence. Intravenous urography will be very helpful in determining whether ligation or complete severance has occurred.

If the accident is discovered within the first forty-eight hours, it may be possible to reopen the wound, locate the area injured and relieve the situation by the cutting of a ligature. Severed ends can be reunited, if located, or the proximal portion of the ureter can be implanted in the bladder provided there is sufficient length. If the situation is not recognized until later, immediate bilateral nephrostomy should be performed. Instances have been reported in which the sutures have been absorbed within a week following the nephrostomy and the urine flow to the bladder restored.

#### Anuria Due to Drug Lithiasis

Drug lithiasis may be appropriate as a term to describe the formation of concretions in the urinary tract following the use of the *sulfonamide drugs*. Sulfapyridine, sulfathiazole, and now sulfadiazine concretions have been noted clinically.

CASE REPORT.—R. W., a white man aged forty-two years, was admitted to the medical service of the St. Louis City Hospital, October 22, 1939, complaining of having had a cold for the previous three weeks. On the day prior to admission he began having intermittent chills, hiccoughs and pain in the chest. The pain was accentuated on coughing and deep respiration. The past history was essentially negative except for an appendectomy in 1930 and malaria in 1933.

On admission the patient, who was well developed and well nourished, was shaking violently and appeared acutely ill. His temperature was 100.4° F., his pulse was 100, and respirations numbered 24 and were shallow. The nasal passages were obstructed, the throat was red, and a postnasal drip was present. The breath sounds were depressed over the left base but otherwise no changes were noted. The heart and the abdomen were normal. The blood pressure was 110 systolic, 75 diastolic.

The erythrocyte count was 3,770,000 per cubic millimeter of blood, and white cells numbered 5600, with 58 per cent seg-

ing the administration of 6 gm. of sulfathiazole daily for four days. The nonprotein nitrogen of the blood rose to 99 mg. per 100 cc. and the sulfathiazole content of the blood to 16 mg. per 100 cc. The pathologic report of the autopsy specimen of the kidneys by Dr. Hollis Allen was:

"Both kidneys are average in size and show a rather marked granularity beneath the capsule. The cortex is average in thickness. The pelvis is very slightly dilated. The wall of the pelvis is somewhat thickened. Both pelves contain a large amount of a light brownish sandy material. In the left kidney the largest grains are about the size of the head of a



Fig. 55.—Sulfathiazole crystals in renal pelvis in fatal case of anuria following sulfathiazole therapy.

match; in the right kidney the largest grains are about the size of the head of a pin. There is rather striking uniformity in the sand in each pelvis (Fig. 55).

"Microscopic examination of the kidneys shows a diffuse arteriosclerotic process with rather marked cloudy swelling. In the capsules of the glomeruli there may be seen an amorphous debris. There is rather marked congestion of the kidneys."

#### POSTOPERATIVE UREMIC SYMPTOMS

The syndrome noted is a diminished urinary output, a thick, red beefy tongue coated white on the surface, a fetid breath, dry skin, drowsiness, hiccoughs and a rising non-

this a rapid, clear urinary drip was obtained on both sides. A subsequent pyelogram showed normal renal pelves except for a small filling defect in the right pelvis.

The patient showed immediate clinical improvement. The urinary output returned to normal following cystoscopy and the nonprotein nitrogen of the blood, estimated October 30, was 27 mg. per 100 cc. The urine two years later continued to show from 10 to 15 red blood cells and from 2 to 3 white blood cells per high-power field.

*Comment and Summary.*—1. The supersaturation of acetyl-sulfapyridine crystals during the administration of sulfapyridine is sufficient at times to cause a complete obstruction in the urinary tract. This same condition has been reported after the administration of sulfathiazole and sulfadiazine. No such formations have been observed following the use of sulfanilamide or neoprontosil.

2. Immediate measures consist in the forcing of fluids, administering a 10 per cent solution of glucose intravenously and discontinuing the drug. If these fail, the condition can be promptly and satisfactorily relieved by inserting catheters cystoscopically in the ureters and pelves, and lavaging them with warm physiologic solution of sodium chloride or sterile water.

3. These crystals, even in large amounts, are not opaque to the x-rays; therefore, a flat roentgenogram is of no value. The symptoms of renal colic and/or hematuria should make one suspicious of crystalline concretions in the urinary tract and should not be mistaken for gastric upsets.

4. The forcing of fluids in conjunction with sulfapyridine therapy is appropriate, since it will relieve the supersaturation of the urine to some extent. Although Flippin<sup>5</sup> et al. reported fewer crystals in alkaline urine, we have not observed any effect in vitro or vivo with the change of hydrogen ion concentration. The crystals will dissolve in a hot solution.

Following the use of the sulfonamide drugs, anuria may also occur as a result of the deposition of crystals throughout the *tubules* and renal *cortex*. Such an instance was seen by us in consultation in September, 1940. A seventy-six-year-old patient, treated for pneumonia, had complete anuria follow-

## Loss of Chlorides and Other Electrolytes; Rechloridization

A whole literature is growing up, following the work of Chabanier and Lobo-Onell, on the disturbance of the chloride equilibrium in the blood and in the tissues following disturbance of the renal function. The observations have been grouped about the preparation for and the prognosis of prostatectomy, but they apply actually to the prophylaxis and the treatment of urinary sepsis in general. Thus Van den Branden summarizes our present knowledge as follows:

"Nitrogen retention almost always follows even the slightest *surgical intervention*. This may lead to an acute renal insufficiency, even to complete anuria.

"After operation the blood chlorides roughly parallel nitrogen retention. Yet the chlorides are not eliminated in the urine for the urine chlorides are simultaneously reduced. Legueu, Fey and Palazzoli found that traumatized tissue shows a rise in chloride content. Hence they conclude that chlorides are drawn to traumatized tissue.

"Duval's researches on postoperative nitrogen retention led him to believe that traumatized tissue liberates toxic or proteolytic substances. Chabanier conceives that these circulating toxins may cause secondary tissue changes at a distance, thus liberating other toxins. If, by chance, the kidney or bowel which normally would take care of these toxins are inefficient, either of themselves or because of toxic inhibition, nitrogen retention, oliguria, anuria, shock, etc., follow promptly, unless the toxins are neutralized in the first place by sodium chloride. Hypochloremia is not itself a cause of renal insufficiency but a consequence of trauma to tissue.

"Actually the clinic confirms this theory. Chabanier and Lobo-Onell have, by intensive rechloridization, dissipated grave symptoms of nitrogen retention and achieved veritable resurrections. Moreover we find that preliminary rechloridization either prevents the formation of toxins or neutralizes them as they form.

"Although chloropenia is not of itself pathogenic, we use it as a measure of the progress of toxemia and of chloride therapy.

"Van den Branden has noted that 'without rechloridization before operation the patient after prostatectomy passes about 300 cc. of urine—whereas with preliminary rechloridization seven out of nine passed more than a liter in the first 24 hours. No postoperative shock. Postoperative nitrogen retention slight.'

protein nitrogen of the blood. The treatment depends on recognition of (1) dehydration, (2) loss of chlorides and other electrolytes, and (3) acidosis.

### Dehydration

The postoperative patient requires an intake of from 2000 to 4000 cc. of fluid each twenty-four hours to replace the amount lost in water through (1) vaporization (800 to 2000 cc.), (2) urine (1000 to 1500 cc.), (3) stool (200 cc.) and (4) vomitus (500 cc.). Coller<sup>6</sup> states that when the patient manifests the symptoms previously noted he has lost fluids amounting to 6 per cent of his body weight. Thus, if an individual weighs 60 kg. he should have 3600 cc. each twenty-four hours. As a patient should normally excrete 1500 cc. in the same period, fluids should be forced until that output is attained. The most frequent cause for deficient excretion in the first twenty-four hours following operation is insufficiency of the fluid intake and not shock to the kidneys. Diminished output causes a high specific gravity. The more damaged the kidney is, the less able it is to handle salts. Therefore, the fluids should be forced so that the kidneys can eliminate all the salts as waste products instead of retaining them as toxic elements. A simple method of calculating the total solids in a liter of urine is to take the last two figures of the specific gravity and multiply them by 2.33 (*Haeser's formula*). For example, if the specific gravity of the urine is 1.010, the total solids excreted in a liter will be 23.3 gm. Since the total solids excreted normally should be around 50 gm., the output obviously should be 2200 cc., in this particular patient.

In general, the lower the specific gravity in a twenty-four-hour collected urine, the greater should be the intake of this individual.

The many methods of administering fluids are well known: by mouth, through nasal tube, by rectum, by hypodermoclysis, or intravenously. Tap water is easily absorbed by rectum. Normal saline solution or 4.7 per cent glucose may be given under the skin. The intravenous route has the widest use, since hypertonic or hypotonic solutions can be given, but it should be selected scientifically.

an alkalinoses, and it has the further advantage in that the proper amount to be given can be fairly accurately calculated from the amount of acidosis present. The following formula has been found to be quite reliable.

"To obtain the number of cubic centimeters of sodium lactate r to be given a patient, subtract the observed  $\text{CO}_2$  content in volume per cent from the normal which is 60 and multiply by 0.3 of the body weight in kilograms.

$(60 - \text{observed } \text{CO}_2 \text{ in volume } \%) \times 0.3 \text{ kg. body weight} =$   
cc. of sodium lactate r.

To make the solution isotonic, add the sodium lactate to 5 times that amount in normal saline solution ( $\frac{1}{6}$  molar). In patients with edema, a  $\frac{1}{6}$  molar solution need not be used but full strength or  $\frac{1}{2}$  may be desirable."

#### BIBLIOGRAPHY

1. Carroll, Kappel and Allen: Sulfathiazole and Its Sodium Salt—Effectiveness and Limitations in General Practice. *J. Urol.*, 45: 1033-1042 (Nov.) 1941.
2. O'Connor, Vincent J.: Prostigmin as Aid in Expulsion of Ureteral Calculi. *Brit. J. Urol.*, 11: 325-331 (Dec.) 1939.
3. Lewis, Bransford: Regurgitation Renal Colic. *Urol. & Cutan Rev.*, June, 1932.
4. Ockerblad, N. F. and H. E. Carlson: Stones of Recumbency. *South. Med. J.*, 53: 582 (June) 1940.
5. Flippin, H. F.: Sulfadiazine and Sulfathiazole in Treatment of Pneumococcal Pneumonia. Progress Report on 200 Cases. *Am. J. Med. Sc.*, 201: 585 (April) 1941.
6. Collier, Fred A.: Studies on the Altered Chemistry of Altered Patients. *Bull. St. Louis Med. Soc.*, 33: 373, 1938-1939.
7. Hartman, Alexis F.: Clinical Studies in Acidosis and Alkalosis. *Ann. Int. Med.*, 13: 940, 1939.

"A simpler method to arrive at the evidence of tissue-destruction-toxemia is perhaps by study of the alkaline reserve as indicated by the  $\text{CO}_2$  combining power of the blood."

The daily intake requirement of sodium chloride is about 5 gm. This amount is contained in about 500 cc. of normal saline solution. The normal plasma chloride is 550 to 600 mg. per 100 cc. When the uremic symptoms described above appear, the plasma chloride should be determined, and, if found deficient, the proper amount of chlorides should be added to the intravenous solutions.

How much chloride should be added? Coller states that for practical clinical purposes the following may be used:

"For each 100 mg. per 100 cc., that the plasma chlorides need to be raised to reach the normal, the patient should be given 0.05 gm. of salt per kilogram of body weight. For example, if a patient weighing 60 kg. is found to have a plasma chloride level of 410 mg. per 100 cc., and it is desired to raise the plasma chlorides to 560 mg. per 100 cc., the sodium chloride need may be calculated as follows:

$$\frac{560 - 410 \text{ mg.}}{100 \text{ mg.}} \times 0.5 \text{ gm./kg.} \times 60 \text{ kg.} = 45.0 \text{ gm. of salt}$$

which would require 5,294 cc. of normal salt solution."

Immediately following an operative procedure there is a marked loss of chlorides amounting to as much as 25 gm. The administration of 2000 cc. of normal saline will provide from 20 to 25 gm. of sodium chloride which is obviously in excess. However, in a short while the daily need is only 5 gm. (500 cc.) of normal saline and prescribing of the remainder of the fluid requirement in the form of 5 per cent glucose in the sterile distilled water is more rational after the first few days.

### Acidosis

For combating acidosis as noted by a low alkaline reserve we have found the sodium lactate as suggested by Hartman<sup>7</sup> to be the most practical. The lactate is more soluble than sodium bicarbonate and, therefore, more easily sterilized. It is more rapidly excreted and thus is not retained to produce

## TREATMENT OF GASTRO-INTESTINAL EMERGENCIES\*

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THE physician is called in the gastro-intestinal emergency because of one or more magnified symptoms from the disturbed gastro-intestinal tract or the abdomen. These symptoms—pain, vomiting, hemorrhage, diarrhea, meteorism, collapse, chill and hyperpyrexia—are often exaggerated beyond any characteristics which they usually have for specific maladies. The physician is immediately faced with the problem of diagnosis, a prompt prognosis and usually the need of surgical consultation. Diagnosis in a definite manner is often difficult.

Gastro-intestinal emergencies involve the differential diagnosis of appendicitis, esophageal obstruction, pyloric obstruction, perforation or hemorrhage of peptic ulcer, acute gastric dilatation, acute diverticulitis, acute cholecystitis, biliary colic, peritonitis, hemorrhagic pancreatitis, acute enterocolitis, poisoning, basal pneumonia, diaphragmatic pleurisy, mesenteric thrombosis, ruptured tubal pregnancy, and intestinal obstruction by internal hernia, localized luminal disease, adhesions, foreign bodies, intussusception and volvulus. Surgical consultation and operative interference are often immediately imperative. The physician finds opportunity in these emergencies for the exercise of the highest diagnostic skill, and judgment in securing adequate and timely surgical care and in preparing and supporting the patient through the emergency. Medical measures are not often direct but are of great importance.

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and also as a means for shortening convalescence. These considerations are valid irrespective of the site of the bleeding into the tract.

As previously stated, the treatment of hematemesis, irrespective of its cause, may initially follow that proposed for chronic peptic ulcer until such time as full diagnostic investigation may be secured. In peptic ulcer the hemorrhage occurs because of active destructive progression of the ulcer, and, beyond the brief period in which the natural physiologic factors of hemostasis are allowed to act and are abetted by complete rest, the treatment of hemorrhage is the treatment of peptic ulcer, by which healing of the ulcer is promoted. The primary measure to that end is the immediate control of the acid gastric secretions which is best achieved by food and antacids and these should be started after the first twenty-four hours of quiet and starvation.

The diet may be liberal and the menus built to absorb the secretions, with a paucity of fluids with the meals. The fluid balance may be maintained by water between meals. The diet may contain cereals, toast or bread and butter, cheese, mush or grits, eggs, meat, puddings and milk. The feedings may be five or three and an *antacid* should be used between feedings. The author has found a combination of calcium carbonate, bisubcarbonate and sodium bicarbonate with calcined magnesia adequate for the bowels, and in every way efficacious. In cases in which there is an obvious or suspected pyloric obstruction, the diet will need to be lessened and often the stomach will need to be decompressed by aspiration. Cessation of bleeding will be revealed by the blood studies, and by disappearance of the guaiac reaction of the stools. The emergency is then past and adequate diagnostic procedure may be safely instituted.

#### PERFORATION OF PEPTIC ULCER

The perforation of peptic ulcers occurs suddenly with atrocious stabbing pain and presents to the patient and physician an urgency seldom equaled by other gastro-intestinal accidents. This severity of onset in itself indicates the probable diagnosis which, in a great majority of cases, is supported by

HEMORRHAGE FROM THE GASTRO-INTESTINAL  
TRACT

Sudden and copious bleeding into the gastro-intestinal tract may be signaled by the vomiting or passage of blood, or both, accompanied by weakness, sweating and thirst, or complete syncope. The episode is rarely fatal but is a major emergency in the course of the primary disease. A slower and continued bleeding, without such spectacular symptoms and revealed only by melena and progressive anemia, may reach an emergency status before it is recognized by the patient. Repeated small transfusions are indicated for hemostatic effect, and essential restoration of blood volume and oxygen capacity.

Copious hemorrhage of little changed blood from the bowels may occur as an initial manifestation in diverticulosis, cancer and polyp of the colon and less frequently in blood dyscrasias. Hematemesis commonly occurs in gastritis, esophageal varicosities and chronic hepatic and splenic disease, as a complication of chronic peptic ulcer, and as an initial symptom in acute, punctate nonpeptic ulcers. From the standpoint of *diagnosis* there is a dangerous tendency to consider too quickly chronic peptic ulcer as the source of hematemesis. Even with a suggestive clinical history of hunger distress, this diagnosis may prove erroneous. It is, however, valid to treat all such cases as for peptic ulcer until diagnostic measures are safe and available.

The treatment of sudden and copious hemorrhage is *complete bed rest*, and *starvation* for a brief period of twenty-four hours. *Sedation* by means of bromide and barbiturates is necessary to combat restlessness and apprehension. Morphine, dilaudid, or pantopon may be used but their tendency to produce nausea makes them treacherous. Very seldom does hemorrhage precipitate a dangerous collapse, and the transient syncope and persistent low blood pressure are desirable factors in hemostasis and are not indications for immediate *transfusion* so long as the quiet patient has no definite cardio-respiratory distress or failure. Only in the infrequent moribund exsanguinated patient should transfusion be employed immediately. After a variable period of one to five days transfusion may be considered both from its absolute indication

passage of the tube will also keep the stomach practically empty but intubation will need to be frequent in the early hours of the condition. Much of the fluid is from the duodenum and the systemic effects are more profound than from merely the loss of the electrolytes of the gastric secretion. A rise in the level of blood nitrogen is significantly prognostic. After the initial evacuation of the stomach the aspiration should be repeated at hourly or longer intervals until the accumulation of fluid ceases. *Feeding* may be started at this time. Liquid nourishment and water may be allowed in small amounts of 4 to 6 ounces at intervals of two to three hours, with further aspiration of the stomach prior to each feeding. As the quantity of fluid aspirated at such times becomes scant, the feeding may be increased in quantity and the intervals lengthened. During the initial period of decompression aspiration of the stomach, saline and glucose, combined or separated, may be administered intravenously. Transfusion of blood or plasma may become indicated.

Treatment of acute dilatation of the stomach, *when initiated promptly*, is usually successful; if initiated late, the patient may already have reached such a stage of prostration that there is little tendency toward recovery, the systemic effects having become irreversible. The muscular compensation of a hollow viscus when distended to the point of exhaustion is prompt after evacuation and with maintenance of the empty state. Few physiologic recoveries are more satisfactory. Prompt recognition of the condition is therefore of determining importance.

#### ACUTE APPENDICITIS

The medical treatment of the acute abdominal attack due to appendicitis is in general negative. The patient is usually in bed by his own choice. Seldom does he remain ambulatory after the onset of symptoms, but occasionally the atypical position of the appendix, especially the retrocecal appendix, will so modify the pain as to allow this. Pain may be the only symptom which disturbs the patient, but nausea and vomiting in association with the pain are usual. If nausea is absent or present only in a minor degree, vomiting is unlikely.

an antecedent history of peptic ulcer. The picture of the acute abdomen with muscle defence and fixation of the diaphragm perfects the diagnosis, which often may be confirmed by the roentgenologic demonstration of a pneumoperitoneum. The position of the air may be ascertained, according to the posture permitted the patient, as subdiaphragmatic or subumbilical. The air may, even in moderate quantity, give a characteristic percussion tympany. A leukocytosis develops promptly. Often shock accompanies the onset; on the other hand, the passing of the first violent pain may mislead the observer, especially as the pulse and temperature may not yet be elevated. The usual characteristic pattern of symptoms and signs may vary when the perforation is posterior, when covered by recent serosal reaction or when the leakage is slow.

In the *differential diagnosis* hemorrhagic pancreatitis and appendicitis, especially when subhepatic, must be considered. The *prognosis* for perforated peptic ulcer depends primarily upon the element of time. Prompt diagnosis and adequate operative interference carry a favorable prognosis.

In most instances the diagnosis is evident and *immediate operation* is imperative. Indecision is dangerous. The operative risk increases rapidly with the hours and becomes unfavorable after a twenty-four-hour delay, although it is very good within a six-hour period. The medical responsibility is circumscribed and prompt surgical attention is urgent.

#### ACUTE DILATATION OF THE STOMACH

The acute dilatation of the stomach occurs suddenly and progresses rapidly to a fatal termination unless it is recognized and treated. It usually follows recent laparotomy or abdominal injury and is characterized by continued, easy, repeated, scant vomiting, indefinite pain or discomfort, and fullness and distention of the epigastric area or even of the whole abdomen, with associated tympany and succussion. Without relief prostration supervenes.

*Treatment* consists in *immediate and repeated aspiration or decompression* of the stomach. The stomach must be kept empty. Aspiration is preferably accomplished by an indwelling nasal or stomach tube with continuous suction. Repeated

major anomalies of intestinal rotation and mesenteric attachment permit the rare massive volvulus upon the root of the mesentery. It is probable that a small, transient, incomplete volvulus occurs frequently, without strangulation of mesentery, but with transient pain and distention.



Fig. 56.—Observation of a barium meal in a case of intestinal obstruction showing as yet few of the effects of obstruction, which however developed rapidly as shown in the following illustration.

*Intussusception* is especially acute and dangerous in the infant and during childhood, being of primary etiology. It is then usually progressive and requires prompt attention. The palpable "sausage" mass and bloody dejections with the obstructive signs complete the diagnosis. Intussusception in the adult is commonly a secondary development upon the basis

It is the physician's primary concern to *prevent medication*, especially purgation, which yet is often an initial and urgent inclination of the patient and family. The prohibition of all food and restriction to scant fluid are essential, until observation has permitted diagnosis. When the diagnosis has been made, operative interference should be prompt. When uncertainty continues it is imperative that early consultation should be had and a decision reached.

The *symptomatic relief of pain* is also interdicted until the diagnosis is made. The spontaneous amelioration of a pain suggesting appendicitis must be observed with extreme caution even when pulse, temperature and other signs have improved. The recrudescence of pain or increased leukocytosis urges immediate interference. Operation within twenty-four hours carries statistically a favorable prognosis but the progress of gangrenous appendicitis is often so rapid that a delay of six hours may change the favorable prognosis to an unfavorable one.

#### INTESTINAL OBSTRUCTION

Acute intestinal obstruction may occur from localized disease, carcinoma, ulceration and adhesions, or from intussusception, volvulus, herniation, and foreign bodies. In general, it exhibits the same symptoms and signs irrespective of cause. A history of previous attacks of disease, or antecedent laparotomy or wounding of the abdomen, point to adhesions as the more probable cause of obstruction. Among diseases which may cause such obstruction without antecedent laparotomy are appendicitis, pelvic cellulitis, colonic diverticulitis, pericolicitis and pericholecystitis. Obstruction as a sequel to such diseases is usually of the small intestine. Colonic diverticulitis may directly and acutely obstruct the colon.

*Volvulus* may occur in the small intestine but is more often of the colon, especially of the redundant distal colon, and less frequently of the ileocecal segment. The degree of redundancy in the pelvic and sigmoid segments of the colon and the narrowness of the mesenteric base determine the liability to volvulus. The presence of a mobile cecum with a free mesentery is necessary for volvulus of the ileocecal segment. Other

obstructions are less fulminating as regards the active vomiting and the loss of fluid and its sequelae. A fecal vomitus is a late sign and its appearance should not be awaited. Obstruction in the colon may be initially without vomiting and without the loss of fluid. Distention will gradually increase, with the outline of the distended intestine or bowel often being visible and exhibiting peristalsis. The loop of a volvulus, especially one of the sigmoid, may be evident on inspection.



Fig. 58.—An acute intussusception with obstruction in a child. The invagination has progressed through the hepatic flexure of the colon. The small intestine is distended with gases.

The acuteness of symptoms may also vary according to the *suddenness of onset* of the actual obstruction, which when suddenly complete will give a violent clinical reaction, whereas a slowly developing obstruction, as in slow stricture of the intestine or bowel by localized disease, may develop a tolerance which is astounding and misleading. The onset of acute obstruction may be with sudden pain, initially local-

of a localized hypertrophic disease and is characterized by repeated attacks of pain, distention and relatively sudden resolution. These anticipating attacks increase in their duration until a major obstruction occurs.

*Primary obstruction of the small intestine* occurs from antecedent ulcerating disease which produces strictures. This disease may be either of specific etiology or an idiopathic regional ileitis. There is often an antecedent history of minor,



Fig. 57.—Observation at sixty-nine hours in the same case as Fig. 56, showing the characteristic dilated intestine of complete obstruction.

transient obstructive attacks, probably from food obstruction of the strictured lumen, until finally a persistent obstruction occurs.

When obstruction of the bowel becomes suddenly complete, *pain, distention and vomiting* are the major symptoms. Vomiting will vary according to the level of the obstruction, which when high, gives rise to urgent, repeated vomiting with great loss of fluid and prompt collapse of the patient. Lower

is essential. Small and frequent feedings may soon be allowed and an antacid in the intervals of feeding will gradually cause the acute tissue reactions to subside. Only then may the acuteness or chronicity of the stenosis be properly assessed and the medical prognosis determined.



Fig. 59.



Fig. 60.

Fig. 59—Observation of the barium meal at six hours in a case of pyloric obstruction from acute exacerbation of a duodenal ulcer. There was scant clearance and the stomach shows marked ectasia.

Fig. 60—The stomach of the same patient, as seen in the initial observation of a barium meal. After twelve days of aspiration and ulcer treatment it shows normal size and its motility was completely restored.

### ACUTE ESOPHAGEAL OBSTRUCTION

Acute esophageal obstruction with its symptoms of dysphagia and clinical history of foreign body occlusion or caustic poison burn is generally of ready diagnosis. In the case of *foreign body*, prompt esophagoscopy and removal are necessary procedures in treatment. Without a conclusive history, a barium fluoroscopic or radiographic examination may establish the diagnosis. Infrequently, the *congenitally dilated esophagus* may become obstructed at the cardia by acute

ized, but later generalized and acquiring the intermittence of peristalsis. There is increasing abdominal distention and inaction of the bowel without the passing of stool or flatus. A roentgenologic observation with vertical and prone films of the abdomen will demonstrate the characteristic fluid levels and distended loops.

Immediate medical treatment entails *decompression of the stomach and intestine* and the intravenous administration of glucose and saline. The latter is important to combat the progressive prerenal increase in blood nitrogen. Mechanical obstruction of the intestine needs prompt surgical relief and decompression can be only in preparation for surgery.

#### PYLORIC OBSTRUCTION

Pyloric obstruction develops usually from cancer or peptic ulcer and occasionally from postpyloric adhesions or pericholecystitis. In any case it may be relatively sudden in onset, although commonly the obstruction is of gradual development and exists as a partial condition. The clinical history and adequate roentgenologic study will in a great majority of cases establish the primary disease. The pyloric obstruction of *cancer* requires prompt surgical attention. The pyloric obstruction of *peptic ulcer*, duodenal or gastric, should be initially treated by medical management. In a large percentage of cases, the obstruction may be corrected and an unnecessary operation avoided. It is therefore important to relieve the obstructed stomach of the immediately embarrassing factors of retention and continuing acidity, and thus to allow subsidence of the acute reaction of the tissues in order to differentiate between chronic and acute obstruction. In the one it will accurately define the surgical indication and present the patient as an improved surgical risk. In the other it will relieve the obstruction and allow the pursuit of the full medical management of ulcer.

In the *lesser* obstructions of ulcer, greatly diminished intake and an antacid may promptly restore adequate gastric motility and size, and soon a liberal, full maintenance ulcer diet may be reinstituted. With *major* pyloric obstruction, decompression of the stomach by continued or repeated aspiration

Among gastro-intestinal emergencies acute diverticulitis is distinguished in that *surgery is contraindicated*. Surgery is reserved for the late complications of the chronic disease. The medical care of acute diverticulitis entails rest preferably in bed, the use of no cathartics, atropine sulfate to the extent



Fig. 61.—Diverticulosis of the entire colon. In this instance diverticulitis in various areas may simulate various intra-abdominal diseases. Medical treatment controlled the inflammation tendency in this case.

of tolerance and warm enemas preferably of magnesium sulfate 0.5 to 1 per cent. For the immediate comfort of the patient the use of codeine is indicated. This may be thought to assist in relaxation of the bowel where the spasm of the muscles tends to close the stomata of the diverticula.

#### INFARCT OF THE MESENTERY

An infarct of the mesentery may be restricted or extensive. It may occur by *arterial embolus* or *venous thrombosis*. The first is precipitate, and the latter more gradual, but in either

esophagitis involving the cardiac orifice, and vomiting from this capacious esophagus may be confused with that of pyloric obstruction. The initial stasis and regurgitation may be induced by the patient, and dehydration and loss of weight eventually result. Decompression and lavage of the esophagus will promptly ameliorate the cardiac orifice obstruction in most instances. Repeated episodes of distal esophagitis may induce a contracture of the diaphragmatic segment.

For *chemical burns* of the esophagus the immediate treatment is with antidotal substances—alcohol for phenol, milk of magnesia or soda for acids, vinegar for lye, and generally milk, eggs and olive oil. Unfortunately, such treatment is seldom possible within the adequate time for significant relief. The patient will need subcutaneous and intravenous fluids. The continued acute dysphagia of stricturing poison burns will require surgical interference by gastrostomy. Subsequently, dilatation of the strictured segment by careful bougienage is carried on. Restoration of the normal function of deglutition can often be secured.

*Cancer* of the esophagus is of insidious development and too often patients postpone diagnosis and care until obstruction due to food blockage or esophagitis more or less suddenly becomes extreme. Roentgenologic diagnosis and divulsion of the stricture by bougienage may maintain natural deglutition until this fatal disease has run its course. Failure of easy accomplishment of bougienage indicates prompt gastrostomy.

#### ACUTE DIVERTICULITIS OF THE COLON

Acute diverticulitis occurs as an episode of inflammation in multiple diverticulosis of the colon. The attacks are characterized by pain, tenderness, fever and constipation and vary in degree and duration. In severe attacks a mass may be palpated at the site of inflammation, and the pain may be such as to cause alarm and to embarrass movement. Constipation accompanies the condition. As diverticulitis may occur in any segment of the colon it may simulate appendicitis and cholecystitis. Its greatest frequency is in the sigmoid where it is more readily recognized. Repeated attacks lead to chronic diverticulitis and pericolitis.

## DIABETIC ACIDOSIS

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SINCE the author has had very little experience with the treatment of diabetic acidosis in children, the following discussion applies to adults only.

### **PATHOLOGICAL PHYSIOLOGY**

#### **Ketosis**

The cause of diabetic acidosis is the excessive production of *ketone bodies*. These are acetone, diacetic acid and beta-hydroxybutyric acid. Of these the parent substance is diacetic acid, and acetone and beta-hydroxybutyric acid have their origin from that substance.

*Origin of Ketone Bodies.*—There has been a great deal of work of late on the production and oxidation of the ketone bodies in the body and some of the old conceptions have been discarded by many physiologists.<sup>1</sup> It is now believed that the ketone bodies have their origin almost wholly in the liver and that, although they are oxidized in many of the tissues of the body, the muscles are the main organs for their disposal. Although in the past the ketone bodies were considered abnormal products of metabolism, it is now believed that they are normal products of the oxidation of fat by the liver.

*Oxidation of Fat.*—Soskin and Levine<sup>2</sup> have lately reviewed the origin of ketone bodies. They discuss the different theories of the oxidation of fat; namely the old beta oxidation theory of Knoop; the multiple alternate oxidation theory of Hurlley and the recent theory of beta oxidation and acetic acid condensation of MacKay. Under the old theory of beta oxidation, one molecule of fat produced one molecule of aceto-acetic acid; while under either of the latter two theories, one molecule of fat gives rise to four molecules of aceto-acetic acid.

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case the clinical onset is with pain, usually most extreme, and this is followed soon by vomiting and later by diarrhea, watery at the beginning but later bloody. There is initially little or no abdominal rigidity, no tympany and no intestinal sounds. Intestinal peristalsis is wholly inhibited. The patient is profoundly ill. In arterial infarction the obstructed bowel and the rapidly developing gangrene dominate the picture. However, the violence of the reaction obscures differentiation. In cases of extensive infarction from venous thrombosis there is a rapidly progressive anemia and failing pulse, rapidly progressing to shock and collapse. There are combined in the clinical picture the evidences of hemorrhage and of obstruction of the bowel. The patient is in fact losing blood by sequestration in the portal lake. From this, the intestinal loss of blood may become very great. Early systemic effects are largely those of hemorrhage.

The patient with mesenteric infarct must be supported by *transfusion*. Intravenous fluids are not sufficient. Only transfusion will carry the patient through the operative interference which is immediately necessary. The operative prognosis depends upon the extent of the mesenteric and intestinal involvement, and is in all cases extremely unfavorable.

in the basal metabolism during acidosis but it is sufficiently common to suggest that acidosis itself increases the basal metabolic rate.

*Production of Ketosis.*—The accumulation of ketone bodies in the blood and in the tissues of the body would depend not only upon their excessive production as outlined above, but also on the failure of the *muscles* to dispose of them. This question has been investigated and at present there seems to be no evidence that the muscles fail to oxidize ketone bodies during ketosis. This means that, although the muscles continue to oxidize the ketone bodies when they are produced in excess, the rate of ketone destruction cannot keep pace with the rate of production.

Let us trace, step by step, the production of ketosis. The first step must be the substitution of fat metabolism for carbohydrate metabolism. This occurs during carbohydrate starvation or when there is a lack of insulin or when the diet is predominantly fat and contains very little carbohydrate. Step two consists of an increase in fat mobilization from the depots of the body, with the result that the liver is stuffed with fat and that the metabolism of fat by the liver is tremendously increased. This fat is broken down in the liver through the route of ketone formation. There is a great increase in ketone bodies in the blood. The muscles take care of as much of the ketone production as they can but the evidence seems to be that the ability of the muscle to metabolize ketones is limited in extent, so that the last step is the failure of ketone breakdown to keep pace with ketone formation, with the result that a great rise occurs in blood and body tissue level of the ketones.

#### Acidosis and Dehydration

Diacetic acid and betahydroxybutyric acid are weak acids. They must be neutralized by base. The vitality of tissues depends on the preservation of a *pH* in the neighborhood of neutrality. Since the acetone bodies must have a *base* to combine with to preserve neutrality they do so at the expense of the base of extracellular water. This base is lost to the body when it is excreted by the kidneys and, although the latter save some of it, much is lost in the urine. The loss of base of extracellular water upsets

The old idea expressed in the clinical literature by the words "fat burns in the fire of carbohydrate" is no longer tenable. In other words, glucose in itself is not ketolytic, that is, fat is not finally oxidized only when carbohydrate is oxidized. One of the experiments which cast doubt on the old idea is finding that when the acetone bodies are injected into the blood stream of normal animals at the same time with glucose and insulin, the acetone bodies do not disappear from the blood more rapidly than when the acetone bodies alone are injected. That this fact is true in the human has been shown by Koehler, Windsor and Hill<sup>3</sup> who have recently injected the acetone bodies into the circulation of normal men and found that they do not disappear from the blood more rapidly after the administration of glucose or of insulin.

The acetone bodies are not produced in large amounts unless the liver is metabolizing large amounts of fat. The liver will metabolize large amounts of fat in the absence of glycogen or in the absence of insulin which promotes the deposition of glycogen in the liver. It is well known that in uncontrolled diabetes, whether produced experimentally or in the human diabetic, the liver is extensively infiltrated with fat. The deposition of large amounts of fat in the liver is brought about by an increased mobilization of fat from the fat depots of the body and its transport to the liver.<sup>6</sup> The result, therefore, of the absence of glycogen in the liver and the change from carbohydrate to fat metabolism in the body as a whole results in an increase of fat metabolism in the liver and an increased fat deposition in that organ.

A further increase of fat catabolism will take place if the metabolic rate of the body as a whole is increased, as occurs during infection or from other causes. This event further augments the fatty acid metabolism in the liver and results in an enormously increased production of acetone bodies.

*Basal Metabolism in Acidosis.*—There is some evidence that acidosis in itself will increase the total metabolism of the body. If uncontrolled diabetics are subjected to basal metabolic determinations we found many years ago that there is an increase up to as high as 30 per cent in the basal metabolic rate. Not all cases of poorly controlled diabetes show an increase

creases the mobilization of fat and the amount of fat being burned in the body. Furthermore, through insulin, glycogen is deposited in the liver and ketone production is sharply decreased. This gives a chance for the muscles to catch up with the amount of ketones they are burning and, as they burn them, base is liberated in the process. But insulin alone will not correct severe acidosis and dehydration. *Body fluids must be replaced* and, with the fluid, base must also be administered. The body has lost a great deal of the sodium normally combined with carbonate and much of the sodium existing in the form of salt. The fluid of choice is *normal saline*.

For many years there has been much discussion as to whether sodium in the form of *sodium bicarbonate* should be administered in acidosis. Joslin<sup>4</sup> has always contended that salt alone in the form of the *normal saline* best corrects the dehydration of acidosis. As has been pointed out previously, sodium chloride is the main electrolyte lost to the body. As a matter of fact, if sodium chloride is administered its sodium can be used to combine with the carbonic acid constantly produced by the body, to form sodium bicarbonate. Chloride is just as essential as base and can be made available only from exogenous sources. Furthermore, the excess of chloride which results from the dissociation of salt can be excreted by the kidney without loss of base in the form of ammonium chloride. Finally, as is well known, sodium chloride is the base of extracellular water and this is the fluid that is lost in dehydration.

However, when diabetic acidosis has existed for a long time, there will be such a great loss of sodium from the body that even with the correction of the diabetic state and in the presence of a normal blood sugar there may exist such a depletion of sodium that the plasma bicarbonate remains low. In this situation, sodium in the form of bicarbonate or better yet in the form of sodium lactate can be administered to bring the level of the sodium bicarbonate of the plasma quickly back to normal.

#### CLINICAL PATHOGENESIS OF DIABETIC ACIDOSIS

*Excessive Carbohydrate Intake and Omission of Insulin.*—Of the last 145 patients with diabetic coma seen by Joslin, one half were in coma because of excessive carbohydrate intake and omission of insulin. If the diabetic patient has been

the electrolyte concentration and to preserve this concentration water is lost and excreted by the kidneys. By this mechanism the concentration of electrolytes is preserved but dehydration results. Therefore it is an axiom that *acidosis always means dehydration* and the two conditions go hand in hand.

The base of extracellular water is sodium in the form of sodium chloride. As this sodium is lost to combat acidosis, the chlorine liberated migrates to the interior of the cells of the body where it is buffered or neutralized by the proteins of the cells. In time it is eliminated in the urine combined with ammonia, a mechanism which saves sodium to the body.

Another route by which chloride is lost during acidosis of diabetes is through *vomiting*, further increasing dehydration.

Still another factor promoting dehydration is the *extreme hyperglycemia* and the large amounts of sugar to be eliminated by the kidney. The kidney can concentrate sugar to about 5 per cent and to eliminate 100 gm. of it 2000 cc. of urine would be necessary.

The sum of all these factors in diabetic acidosis is the development of extreme dehydration of tissues and blood concentration. One can understand why a state of shock is seen so often in severe diabetic acidosis, for dehydration is an important cause of shock.

*The Effects of Acidosis on the Kidney.*—When one examines microscopically the kidneys of patients dying of diabetic acidosis it is noted that there is very marked cloudy swelling of the renal epithelium. As acidosis progresses the kidney suffers from dehydration to such an extent that it begins to show a failure in its ability to excrete sugar and the acetone bodies as well as the normal constituents of the urine. The author and others have shown that there are cases of diabetic acidosis which show very little sugar in the urine and also very little of the acetone bodies, although the blood may show extreme degrees of hyperglycemia and concentration of the acetone bodies. The failure of the kidney is shown by the rise in the nonprotein nitrogen of the blood and this is an extremely grave sign, for the failure of the kidney means the failure of the most important organ of the body in the effort to combat acidosis.

*The Correction of Acidosis and Dehydration.*—Large doses of *insulin* will reverse the process of excessive fat metabolism and substitute carbohydrate metabolism. This immediately de-

sary to give larger amounts of insulin to counterbalance this effect and maintain glycogen formation in the liver.

Since the diabetic may be expected to live out almost a normal expectancy of life, he may have any form of infection that normal individuals experience. There are, however, certain infections that are peculiarly apt to occur in diabetics. These are *staphylococcus infections* of the skin, either carbuncles on cellulitis; *infectious gangrene* of the lower extremities and infections of the *urinary tract*. All of these occur most frequently in patients who have uncontrolled glycosuria. In a series of 100 diabetics followed by myself for ten years or more, it was very evident that infections occurred much more frequently in those who had uncontrolled diabetes than in those who were sugar-free.

*Other Precipitating Factors.*—Other conditions which may precipitate diabetic acidosis can be briefly mentioned. The *vascular accidents* that are so common in diabetics may be an important factor in this respect. Vascular accidents of the brain do not commonly rapidly precipitate acidosis but coronary infarction may do so, particularly if it is associated with impairment of the circulation. Anoxemia from this cause may produce rapid mobilization of glycogen from the liver and the decrease in circulation of blood through the pancreas can easily reduce the amount of endogenous insulin that the islet cells can produce. *Vomiting and diarrhea*, from any cause, may produce serious dehydration on their own account and augment the dehydration of diabetic acidosis. *Cholecystitis*, particularly when associated with toxic manifestations, is a common cause for the exacerbation of the diabetic state. In the summer months when the temperature is extremely high the loss of salt through *sweat* must be carefully controlled in all diabetics. Finally, in the goiter belt *hyperthyroidism* is not an uncommon cause for the precipitation of diabetic acidosis. Wilder<sup>5</sup> finds that 5 per cent of the cases of severe diabetic acidosis at the Mayo Clinic are due to a hyperthyroidism, and our experience in St. Louis coincides with this statement.

*Summary.*—The pathogenesis of diabetic acidosis is based on an uncontrolled diabetes particularly where insulin has been discontinued. In addition, if there is a toxic infection,

carefully instructed as to the dangers of going on carbohydrate sprees and if he is told emphatically that he must not give up insulin without consulting his physician, most severe cases of diabetic acidosis will not occur. The truth of this statement is evidenced by the fact that diabetic acidosis is extremely rare in physicians. Ignoring the diet and stopping the use of insulin as a cause of diabetic acidosis is preventable and when the diabetic is consistently under the care of a physician, uncomplicated diabetic acidosis should never occur. There is a curious belief on the part of diabetics that when it is impossible to take food, insulin should be omitted. Usually the omission of food is due to nausea or vomiting and the conditions that cause such a state are associated with a greater need for insulin. Occasionally it is the physician himself who has advised the patient to stop taking insulin without having checked the urine for sugar!

*Infections.*—Infections are the next most frequently observed cause for precipitating the state of acidosis in diabetics. If the diabetes is uncontrolled this occurs with greater rapidity than when the diabetes is well controlled. The infection that precipitates acidosis is usually severe enough to cause fever and toxic manifestations.

What is the mechanism by which infections bring about acidosis? In the first place there is an increase in total metabolism when the body is invaded by infectious agents. This means an increase in the breakdown of foodstuffs and a need for increased insulin production. Failure of insulin production means increased ketone production. In the second place, doses of insulin which control the patient in his normal state are not at all sufficient to control the diabetes in the presence of toxic infection. It is a common observation that insulin dosages must be tripled and sometimes quadrupled in the presence of infection. The cause for this need of an increase in the amount of administered insulin may be due to the suppression of insulin, produced by the patient's own islet cells. Furthermore, it is possible that the infectious toxins so affect the metabolic processes within the cell that the efficiency of insulin is very much decreased. Finally, we know that the toxins of infections deplete liver glycogen and therefore it is neces-

and 40 per cent were fully conscious. It is therefore essential to emphasize that *even in the presence of severe diabetic acidosis the patient may be quite conscious and alert*. Joslin uses the term "coma" to mean those cases in whom the carbon dioxide content of the blood plasma is below 20 volumes per cent regardless of the patient's state.

The severity of the acidosis is evident when the *blood pressure* falls to extremely low levels, and the *pulse* becomes very rapid. It is essential to carefully record and watch the blood pressure at frequent intervals in all patients in diabetic acidosis.

*Prognostic Criteria.*—The seriousness of any case exhibiting acidosis can be estimated from six criteria. The prognosis of acidosis is progressively worse with (1) the length of time the patient has been in acidosis as evidence by the history, (2) the degree of stupor, (3) the lowering of the blood pressure to critical levels, (4) the older age of the patient, (5) the heightened nonprotein nitrogen of the blood and (6) the seriousness of the complications that may be present. A patient deeply in stupor with low blood pressure, heightened nonprotein nitrogen and serious complications exhibits the features of impending disaster and in such cases, even with the best of treatment, one out of three die. Since it is a fact that the longer the patient is in acidosis the poorer the prognosis for ultimate recovery, it behooves the physician to work rapidly, be in constant attendance at the bedside and set himself a time limit of *not more than twelve hours* to bring his patient out of acidosis.

#### MANAGEMENT

The management of a case of diabetic acidosis can be simply told by following a case as it is first seen at the patient's home and later treated at the hospital. When impending or established acidosis is recognized in the home, immediate hospitalization is clearly indicated. First, however, insulin is given in a dose of 50 to 100 units. (Every physician should carry insulin habitually.) If the patient is drowsy with signs of fairly advanced acidosis, 100 units is the better amount; especially is this true if typical air hunger is present. If the

in the presence of nausea and vomiting we have all of the conditions necessary to precipitate acidosis. Emphasis should be given to the fact that acidosis in diabetes can develop with extreme rapidity. We have seen diabetics sugar-free in the afternoon or evening and show well developed acidosis when seen the next morning.

#### CLINICAL ASPECTS OF DIABETIC ACIDOSIS

It is hardly necessary to emphasize the need for the early recognition of diabetic acidosis. There is only one way to accomplish this and that is to test the urine for acetone bodies whenever there is a large amount of sugar in the urine. In our opinion the best method is the *ferric chloride test*. It must be remembered that this test is given by salicylates but, when the urine is boiled after adding the ferric chloride reagent, in the presence of the acetone bodies the color will fade and an odor of acetone is noted. When, however, the color is due to salicylates, boiling intensifies the color and there is no odor of acetone. The treatment of mild acidosis is easily accomplished by returning the patient to his diet and for a period of time increasing the doses of insulin.

When acidosis is definitely established the *dry, cool skin* is noticed by the observer and especially the *very dry tongue*. The history of *diuresis* is obtained as is the recent *rapid losses in weight*. There may be noted also a slight but definite increase in the depth of respiration. Those who have adept noses may be able to note the *odor of acetone* on the breath.

With the development of severe acidosis the *air-hunger* type of breathing first noted by Kussmaul is very striking. The patient complains bitterly of *abdominal pains* and it is often surprising to note that this feature of diabetic acidosis is often mistaken as evidence of a surgical condition. Often, however, the pain is not in the abdomen but is referred to any part of the body. *Vomiting* is frequent and adds to the dehydration from which the patient is already suffering.

Diabetic acidosis is usually called "diabetic coma" but this is a misnomer. In 452 cases of diabetic "coma" that Joslin summarizes, 18 per cent of the patients were totally unconscious or in true coma, 40 per cent were drowsy or semiconscious,

cipitated it? In the latter case the physician must not only treat the acidosis but the complicating condition as well.

### Insulin Dosage

It is my conviction that the slow-acting forms of insulin such as protamine zinc insulin are not desirable in diabetic acidosis; however, both Wilder and others recommend their use. To me it seems important to use the fastest-acting insulin available. Subcutaneous injection is the method of choice unless the circulation, as evidenced by a very low blood pressure, is so poor that intravenous administration is indicated. I have already suggested an initial dose of 100 units of regular insulin. This large dose is indicated, in my opinion, in order to promote the most rapid possible delivery of the patient from his acidotic state. Joslin and others suggest smaller hourly doses of insulin. To this I would agree provided the insulin is administered at a constant rate, that is, in doses of the same size at hourly intervals. The physician can then judge, when later data are available, how the rate of insulin administration should be changed. If doses varying in size are administered, it is difficult to gauge the need for change in the dosage as evidenced by the change in urinary and the blood sugar levels.

*If blood sugar determinations can be made at any time of day or night* (as should be the case in any grade A hospital) the physician may wait two and a half or three hours after the large initial dose to get the second blood sugar determination, and thus be able to gauge the size of the second dose of insulin from the effects of the first. The initial blood sugar level in diabetic acidosis is usually in the neighborhood of 500 mg. or more per 100 cc. If at the end of two and a half hours this level has not been lowered by the first 100 units of insulin, then it is evident that another 100 units are clearly indicated. On the other hand, if the blood sugar level has fallen, say, from 500 to 350 mg. per 100 cc., then the second dose of insulin may be from 50 to 60 units. In the event that the blood sugar has actually risen then the second dose of insulin should be greater than the first. After the administra-

patient must go a considerable distance involving several hours of travel, it is essential to administer normal saline intravenously before he is sent on his journey, in quantities of 1000 or 1500 cc.

It is important that the patient be kept warm during transportation to the hospital, which should have notice of his coming so that saline, insulin, nurses and interns will be ready on his arrival. At the hospital the urine and blood should be examined immediately for sugar and the blood for its carbon dioxide content. The urine should also be tested for acetone bodies and examined for bacteria or white blood cells in the sediment. If the initial insulin dose was not given previously then it should be administered immediately, without waiting for the report of the blood sugar value. The injection of intravenous saline and subcutaneous saline should be started without delay.

With these initial procedures under way, the physician can turn to the patient and his relatives for further information regarding the mode of onset of the acidosis. It is important to find out how long the patient has had diabetes, what have been the late events in the course of this disease, how long he has been passing large amounts of urine, whether he has had fever, been vomiting, or had diarrhea. An effort is made to determine the length of time the patient has been in acidosis by ascertaining the duration of the typical air-hunger breathing and the extreme thirst, for what period he has appeared to be rapidly losing weight, and how long he has complained of pain. If these symptoms, particularly those of severe acidosis, have been present for twenty-four or more hours, the physician may be sure he is dealing with a severe case of diabetic acidosis.

Next in order is a careful *physical examination*, keeping in mind the search for infection, evidences of vascular accident, coronary infarction, cholecystitis, enteritis, appendicitis, pyelitis and hyperthyroidism, any of which may precipitate diabetic acidosis. It is well, perhaps, that the physician classify the acidosis as to etiology. Is it due primarily to neglected diabetes or is it secondary to complications which have pre-

cases of diabetic coma. The average insulin dosage in the first twenty-four hours of treatment has varied from 150 to 250 units in these years, and the average for all the years is 205 units.

The condition of the patient during treatment is of very great importance. For instance, a decrease in the intensity of the Kussmaul breathing or the return of consciousness or increased mental acuteness is favorable, but if drowsiness and mental stupor persist after the patient is sugar-free or almost sugar-free the physician should always consider the possibility that the mental condition of the patient is due to some other cause than diabetic acidosis. He should look for evidences of cerebral hemorrhage, meningitis, septicemia, uremia, or extensive liver disease.

The insulin dosage *after the control of the blood sugar* will depend upon the amount of food the patient receives and the severity of diabetes. The amount of insulin that has been required to bring the patient out of the acidosis will be a rough index of the severity of the diabetes. As soon as he can take it without nausea or vomiting, the patient should receive 150 to 200 gm. of carbohydrate each twenty-four hours. With this amount of food, the physician can safely administer at least one half of the amount of insulin that was required to control the diabetic acidosis.

### Fluids

*Saline Solution.*—The loss of fluids in diabetic acidosis can be estimated at from 3000 to 5000 cc. Hartmann finds that diabetic acidosis in children requires 100 cc. of fluid for each kilogram of body weight. We have already recommended the immediate injection of 1000 cc. of *normal saline solution* intravenously and at the same time the starting of subcutaneous injection of normal saline solution. As long as there is rapid absorption of the subcutaneous fluid we believe that continuous administration should be continued. When the absorption becomes slower it can be stopped, provided 1500 to 2000 cc. have been administered. *Ringer's solution* may be used in place of saline solution if it is quickly available.

*Alkaline Solutions.*—The arguments for the use of saline

tion of the second insulin dose, another wait of two to three hours and a third blood sugar determination will indicate the size of the third dose of insulin. In rare instances 200 or 300 units of insulin have been found to have no effect on the blood sugar level. In this event the physician should not hesitate to double and treble the maximum doses of insulin, for there are cases cited in the literature which have required as much as 2000 units of insulin in the first twenty-four hours of treatment.

If *blood sugar determinations are not available* the physician must depend on the amount of sugar in the urine, the intensity of the ferric chloride reaction and the clinical condition of the patient. Although the use of a retention catheter is usually not indicated and is perhaps contraindicated when blood sugar determinations are available, in the situation where one must depend on the amount of sugar in the urine such use is imperative. With the retention catheter in place, the urine can be tested for sugar every hour. In the event that a quantitative determination of the sugar in the urine is not available, one can use as an index of the sugar content the number of drops of urine that will completely reduce 5 cc. of *Benedict's qualitative reagent*. When one or two drops of the urine completely reduce 5 cc. of Benedict's reagent, it is evident that the urine contains large amounts of sugar. The intensity of the *ferric chloride reaction* is very helpful and one is encouraged when the amount of color steadily diminishes, but one should remember that it is entirely possible for the level of the sugar in the urine to decrease steadily while the acetone bodies of the urine remain unchanged. As long as a few drops of urine reduce 5 cc. of Benedict's solution completely, the physician may continue to use large doses of insulin.

In these instances in which the size of the insulin dose must be determined solely by studies of the urine, the insulin should be given at *hourly* intervals, and in quantities of 25 to 50 units. Just as soon as there is evidence that the amount of sugar in the urine is decreasing, the dose of insulin should be halved.

From 1923 to the first of January, 1940, Joslin treated 463

the glucose. The experience of the author is that in the usual acidosis at least 1 unit of insulin should be given for each gram of sugar given intravenously, and in severe acidosis  $1\frac{1}{2}$  units for each gram of glucose. The insulin may be given subcutaneously a half hour before the glucose is begun or, if one prefers, it may be added directly to the glucose solution. I recall a case of intestinal obstruction in a diabetic who was maintained on twice daily injections of 1000 cc. of 5 per cent glucose in saline for a period of seven days. During this time there were only traces of sugar in the urine.

### COMPLICATIONS

*Low Blood Pressure.*—Hypotension calls for the administration of 10 per cent glucose solution, or better yet transfusions of whole blood. The author has very little faith in drugs in this condition; however, 1 cc. of a 1:1000 adrenalin solution may be added to the 10 per cent glucose solution. One ampule of coramine alternating with  $7\frac{1}{2}$  grains of caffeine sodium benzoate every two to four hours may be tried.

*Failure of the Kidneys.*—If the nonprotein nitrogen of the blood is elevated to the neighborhood of 100 mg. per 100 cc., intravenous and subcutaneous fluid should be continued until the level of the nonprotein nitrogen begins to fall. If anuria occurs, 100 cc. or more of 10 per cent salt solution may be tried, especially if the blood chlorides are low.

*Dilatation of the Stomach.*—In our experience, the vomiting of diabetic acidosis stops as soon as intravenous and subcutaneous fluids have been begun. We do not institute gastric lavage as a routine. In the event of continuous vomiting of considerable quantities of fluid, the danger of aspiration pneumonia clearly indicates the necessity for gastric lavage. The stomach may be washed with saline or, as Wilder suggests, with 5 per cent sodium bicarbonate solution.

### SUMMARY

The principles of treatment of diabetic acidosis as recommended in this clinic are:

1. The fearless use of large doses of insulin.

rather than sodium bicarbonate solutions have already been emphasized. Those of us who gave massive doses of bicarbonate in the days before insulin was available, know that it is useless by itself in the treatment of diabetic acidosis. Repeated reference has also been made to the fact that those patients with diabetic acidosis who have been in acidosis for long periods of time may show a failure in the rise of the plasma bicarbonate level even when the diabetic condition is fully under control. In this event the administration of alkali is fully justified. The best form for its administration is *Hartmann's fortified lactate Ringer's solution*. This is prepared by adding 60 cc. of a one-sixth molar solution of sodium lactate to 40 cc. of Ringer's solution, and 500 to 1000 cc. is injected intravenously. Hartmann prefers to administer, without the use of saline, 100 cc. of this solution per kilogram of body weight at the onset of treatment.

If *sodium bicarbonate solution* is the only one available, then a 5 per cent solution to the amount of 500 cc. may be administered. This is prepared by adding to 1000 cc. of doubly distilled water 50 gm. of chemically pure sodium bicarbonate. The water should be first sterilized by boiling and then cooled to at least 70° C. The solution cannot be boiled.

*The Use of Glucose Solution.*—In the author's opinion there are two conditions that may arise during diabetic acidosis in which glucose should be given intravenously. (1) After the blood sugar has been satisfactorily controlled and the sodium bicarbonate of the blood plasma is above 40 volumes per cent, if the patient is still nauseated it may be necessary to administer glucose intravenously instead of giving food by mouth. Such a situation may arise in an infection of the gastro-intestinal tract; *e. g.*, acute cholecystitis. In fact, any condition which prevents the patient from taking food after acidosis is fully controlled, is a clear indication for the use of 5 per cent glucose in saline intravenously. (2) If the systolic blood pressure is rapidly falling, especially below 100 mm. of mercury, the circulation may be sustained at least for a period of time by the injection of a 10 per cent solution of glucose. pending the typing of donors for blood transfusion. The question then arises as to how much insulin is needed to "cover"

## ADDISON'S DISEASE: DIAGNOSIS AND TREATMENT\*

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THE striking features of the clinical syndrome of chronic adrenal insufficiency have naturally directed considerable attention toward this comparatively rare malady. Medical interest in the disease is not in proportion to its importance as a cause of disabling or fatal illness in the general population, but is much greater, because of the secrets gradually being yielded by the study of that mystifying glandular organ, the adrenal cortex.

*Incidence.*—Sir William Osler is said to have observed only seventeen cases in the United States. Up until four years ago, among 150,000 admissions to Barnes Hospital, thirty-six patients were diagnosed as having Addison's disease, and nine others, apparently less typical, were classed as suffering from adrenal insufficiency. As often happens, an awakened interest in a particular problem has led to an apparent great increase in the local incidence. During the past four years in the Metabolism Division of Barnes Hospital and in the Washington University Endocrine Clinic we have personally studied and treated twenty cases of chronic adrenal insufficiency. Of these, thirteen patients have shown rather classical symptoms, six are apparently early cases who have not yet developed a complete clinical picture, and one followed operation upon the pituitary gland for acromegalic gigantism. Three of the patients have died, and in each the diagnosis was confirmed at autopsy.

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2. A time limit of six to twelve hours for the control of acidosis and dehydration.

3. The use of intravenous glucose only after the control of hyperglycemia, when carbohydrate cannot be taken by the mouth, or in the event of a failing circulation.

4. The use of sodium lactate when a low level of sodium bicarbonate in the plasma persists following the control of hyperglycemia.

#### BIBLIOGRAPHY

1. Annual Review of Biochemistry, 1938: Verzar, F., pp. 175-176; 1939: Charkoff, I. L. and Kaplan, A., pp. 225-226, Sperry, W. M., p. 238; 1940: Channon, H. J., pp. 240-242; 1941: Eckstein, H. C., p. 184, Cori, C. F. and Cori, Gerty T., pp. 167. Annual Reviews Inc., Stanford University P.O., Cal.

2. Soskin, S. and Levine, R.: Origin of Ketone Bodies from Fats and their Regulation. *Arch. Int. Med.*, 68: 674, 1941.

3. Koehler, A. E., Windsor, E. and Hill, Elsie: Acetone and Acetoacetic Acid Studies in Man. *J. Biol. Chem.*, 140: 811, 1941.

4. Joslin, E. P., Root, H. F., White, Priscilla and Marble, A.: Treatment of Diabetes Mellitus. Lea & Febiger, 1940, pp. 366-400.

5. Wilder, R. M.: Clinical Diabetes Mellitus and Hyperinsulinism. W. B. Saunders Co., Philadelphia, 1940.

6. Bloor, W. R.: Fat Transport in Animal Body. *Physiol. Rev.*, 19: 557, 1939.

metastases to both adrenals from carcinoma or sarcoma is rare. Other rare causes include hemorrhage, thrombosis, embolism, gummata and amyloid degeneration.

The age at onset is usually between thirty and fifty. Men are more frequently affected than women.

### PATHOLOGICAL PHYSIOLOGY

As the result of the diminution in functional adrenal cortex tissue, the endocrine secretion of its cells is so decreased that serious disturbance is evident in a number of the body's metabolic processes. Complete absence of adrenal cortical secretion results in death. Partial insufficiency may for some time be compatible with life but certain serious functional disturbances occur. These are concerned with:

1. Membrane permeability and electrolyte metabolism.
2. The activity of tissues with specialized functions, such as the kidney, the liver, muscle, and the gastro-intestinal tract.
3. The activation of enzymes.

Present evidence indicates that probably all of the manifestations of Addison's disease can be explained by derangement from the normal in these three categories. It seems likely that the disturbance in membrane permeability, fluid distribution and water balance is primary. Recent investigations have shown that there is not a single hormone secreted by the adrenal cortex, but that there are a number of active endocrine principles furnished by this gland. These principles seem to have various functions, some understanding of which is necessary for a conception of the pathological physiology of adrenal deficiency and its therapy.

Crude adrenal cortex extract can be fractionated into three parts, according to Kendall.<sup>1</sup> These are:

1. Those compounds more soluble in benzene than in water. In the benzene are found corticosterone and dehydrocorticosterone, compound H, desoxycorticosterone, progesterone and other compounds.

2. In the aqueous solution: certain steroid derivatives containing five atoms of oxygen (compounds C, D, E, F, G and others). These can all be removed through crystallization from chloroform.

3. A fraction obtained which is soluble in both chloroform and water, but has not yet yielded crystalline material. This is called the "amorphous" fraction.

*Membrane Permeability.*—When insufficient adrenal cortex

*Definition.*—It would be difficult indeed to improve upon the description of the clinical characters of the disease as given by Thomas Addison in his paper "On the Constitutional and Local Effects of Disease of the Suprarenal Capsules," published in 1855. The following paragraph, taken directly from this paper, is so accurate and complete that it is given as the definition of the disease by a widely used textbook of medicine.

"The leading and characteristic features of the morbid state to which I would direct attention are anemia, general languor and debility, remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of color in the skin, occurring in connection with a diseased condition of the 'suprarenal capsules'."

And later he says ". . . we discover a most remarkable, and so far as I know, characteristic discoloration taking place in the skin, . . . this pervades the whole surface of the body, but is commonly most strongly manifested on the face, neck, superior extremities, penis and scrotum, and in the flexures of the axillae and around the navel. It may be said to present a dingy or smoky appearance, or various tints or shades of deep amber or chestnut brown."

#### PATHOGENESIS AND ETIOLOGY

It is generally stated that, in more than 80 per cent of cases, *tuberculosis of the adrenal glands* is present, with destruction, caseation, and sometimes calcification and fibrosis. Collected statistics on newer groups of cases seem to indicate that, with the decreased incidence of tuberculosis of the lungs and other organs, adrenal tuberculosis is also less common. Simple atrophy and fibrosis account for approximately half of some reported series.

In certain instances the adrenal tuberculosis is the only discoverable tuberculous lesion. More often it occurs in association with pulmonary tuberculosis, or involvement of the genito-urinary tract, lymph nodes, or bones. Among coincidental osseous lesions, tuberculosis of the vertebrae is most common.

Destruction by tumor tissue such as may occur following

All of these physiological disturbances result in a syndrome seen in severe adrenal cortical failure consisting of decreased blood volume, dehydration, low blood pressure and signs of shock, lowered blood sodium, chloride and sugar, and elevation of blood potassium, blood urea and nonprotein nitrogen.

### SYMPTOMS AND SIGNS

The clinical course is variable, but in most cases can be divided into three parts:

1. The *early stage*, lasting for months or years, characterized by asthenia, fatigue, weight loss, anorexia and intermittent gastro-intestinal disturbances, such as nausea, vomiting and diarrhea. Pigmentation may be present for years before the asthenic symptoms become pronounced, or conversely, the change in skin color may be late.

2. The *fully developed*, clinically unmistakable picture of skin pigmentation, asthenia, gastro-intestinal irritability and low blood pressure. The color of the skin is brownish, brownish-gray or beige and occasionally is almost black. The lips, tongue and buccal mucosa may show patchy brown areas, and the nipples and areolae become quite dark. The pigmentation results from an increase in the skin content of melanin, and is darkest where the skin is exposed to sunlight. It is increased in areas of pressure from clothing, in folds such as the axillae and groin, and about scars. Very dark brown or jet-black freckles appear. Frequently the distribution of the pigment is patchy and there may be areas of depigmentation. A feature not emphasized in previous descriptions of cases, but striking in our series is that the *hair* in many patients is prematurely gray or white. In the second stage fatigue on exertion is apt to be so extreme that normal activity is no longer possible. Anorexia may become extreme and there is progressive weight loss. Males lose potency and women become amenorrheic. Abdominal pain with or without constipation or diarrhea is frequent, and acute abdominal states such as appendicitis may be simulated. Headache, light-headedness or dizziness, backache, sensitivity to cold and dyspnea are common. Psychic disturbance is frequent and may consist only

hormone is available, membrane permeability is increased. As Hartman<sup>2</sup> and others have shown, there is more water taken up by muscle from hypotonic solutions, and more water leaves the muscle in hypertonic solutions than in normal animals. Water is absorbed by the cells from interstitial fluid more rapidly than normal. The blood capillary walls allow more fluid to pass through, the blood volume falls, and the blood pressure decreases for this and other reasons. The kidney tubules fail to absorb sodium and chloride ions, but permit increased absorption of potassium. The body loses sodium, chloride and water, there is a fall of these electrolytes in the blood, and hemoconcentration occurs. Desoxycorticosterone and the amorphous fraction of the adrenal cortex have the greatest effect upon the distribution and excretion of inorganic ions. These compounds are a necessary part of the highly efficient mechanism concerned with the transfer of inorganic ions between tissues and fluids and from the blood to the urine.

*Tissue Function.*—Carbohydrate absorption is reduced in adrenal insufficiency (probably because of intestinal tissue derangement), and the ability to form carbohydrates from noncarbohydrate sources is diminished or abolished. The glycogen content of the liver of fasting rats is depressed almost to zero after adrenalectomy. The administration of an unfractionated extract of the adrenal cortex causes prompt deposition of glycogen in the liver. Compound E has the most marked effect upon the carbohydrate metabolism, while corticosterone and 11-dehydrocorticosterone have closely similar action. These compounds are similar chemically in that they all have an atom of oxygen on carbon 11. The amorphous fraction and desoxycorticosterone do not affect the deposition of glycogen in the liver, and have but slight effect upon glycogenesis.

*Enzyme Activation.*—The administration of corticosterone and related compounds is followed by deposition of glycogen in the liver. This result involves the activation of enzymes by cortical hormones. Conversion of protein to dextrose and the formation of ketone bodies in the liver are examples of enzyme action influenced by corticosterone. The deaminization of amino acids by kidney tissue is impaired in adrenal insufficiency. Enzyme activity is increased by giving adrenal extracts or desoxycorticosterone and this function of the kidney may thus be returned to normal. The brownish pigmentation of the skin may be the result of disturbed enzyme action.

ment is started at once. He is covered with blankets to preserve body heat. *Physiological saline solution* is given intravenously with 10 per cent glucose. To this is added from 20 to 40 cc. of the most potent *adrenal cortex extract* available. Care must be taken not to give the intravenous fluids too rapidly. During the first twenty-four hours from 4 to 6 liters of 10 per cent *glucose* in saline solution are given intravenously, and from 40 to 100 cc. of *adrenal cortex extract* is administered intramuscularly or intravenously. When the crisis is not too profound, consciousness is regained within a few hours after therapy is instituted, and the patient may feel remarkably well within twenty-four hours, and may be able to take food and water.

Great caution is necessary at this stage. The patient must be kept flat *in bed* and treatment must be continued, since relapse is apt to occur. The intravenous salt, water and glucose, as well as the hormone injections, are *slowly* decreased in amount and frequency, not suddenly withdrawn. The daily amount of physiological saline is reduced successively to 4, 3 and 2 liters, and may then be omitted, as the patient is able to begin taking added *salt by mouth* in amounts of from three to six 1-gram enteric-coated tablets per day. Intravenous glucose may be omitted when the patient is able to take adequate amounts of carbohydrate foods. The *adrenal cortex extract*\* is reduced gradually as improvement occurs. Maintenance therapy may be continued with from 2 to 10 cc. of the extract daily. It is necessary to choose the extract carefully, since some of the available extracts are of relatively low potency, and the most potent preparation possible is desired.

As the patient recovers from the crisis, it may be advisable to begin the administration of from 10 to 40 mg. of *desoxycorticosterone acetate* daily. This hormone, the only fraction of the adrenal cortex complex made synthetically, and available for therapeutic purposes, is given intramuscularly in oil. It is especially indicated when the blood volume, the blood pressure and the serum sodium are low and the potassium high.

\* The adrenal cortex extract used in our series of cases was supplied by Dr. David Klein of the Wilson Laboratories, Chicago.

of irritability and apprehension, or may be evidenced by delusions, disorientation or noisy delirium.

3. The symptoms described in the first two stages may have been present for months or years, when suddenly more extreme prostration occurs, vomiting appears and the patient is unable to rise from his bed. He is then said to be in "*Addisonian crisis*." He becomes comatose or semicomatose. The blood pressure is often unobtainable or the systolic pressure is 80 mm. or less. The clinical picture of shock is present. The cheeks and eyes are sunken, the skin cold, pale and dry. The heart beat is feeble and rapid and the radial pulse may be imperceptible. The temperature is variable, sometimes subnormal, sometimes very high, particularly if the patient is in extremis. Acetone may be detected on the breath, and the tongue is dry. The crisis may be precipitated by an upper respiratory infection, overexertion, surgical procedure, purgation, salt deprivation or excessive potassium intake.

#### TREATMENT OF ACUTE ADRENAL INSUFFICIENCY

No medical condition is more serious or urgent than acute adrenal failure. The clinical picture will usually make the diagnosis clear. Time will not permit waiting for laboratory reports before starting treatment. It is advisable to withdraw blood enough for chemical studies when the first venipuncture is done for the purpose of giving saline and glucose solution. In many cases no striking *blood chemical alterations* are found, even in crises. The following table will, however, indicate the type of change which may occur:

	Milligrams per 100 cc. of blood	
	Normal	Addison's Disease
Sodium	315-330	260
Chloride (NaCl)	570-620	440
Potassium	16-22	26
Sugar (fasting)	70-100	50-60

The patient is put to bed quietly and quickly. He is moved about as little as possible and the physical examination is limited to the minimum necessary for diagnosis. He must be protected from overzealous friends, family and interns. Treat-

dose of sodium chloride is reduced to 4 gm., it may be necessary to increase the desoxycorticosterone to 5 mg. daily.

The synthetic hormone seems to be less effective in some patients than the complete extract, as far as complete relief of symptoms is concerned, but desoxycorticosterone is much more effective in maintaining weight and blood pressure. Overdosage with the synthetic fraction may lead to excessive increase in blood volume and may precipitate hypertension and congestive heart failure. When the blood pressure exceeds 130 systolic or 90 diastolic, when headache occurs, or edema, or excessively rapid weight gain, the salt dose, the desoxycorticosterone dose, or both should promptly be reduced. Low potassium diets should not be used in conjunction with desoxycorticosterone therapy, since an excessive fall in blood potassium, with marked muscular weakness and prostration may be precipitated.

Hard sterile pellets of desoxycorticosterone may be implanted *subcutaneously*<sup>4</sup> and are absorbed slowly over long periods of time. A number of our patients have been well controlled by pellet implantation, with or without added salt intake. The pellets weighed from 50 to 100 mg. each, and were absorbed at rates of 0.3 to 0.9 mg. per pellet per day. From four to six pellets have proved necessary to keep the blood pressure and weight at normal levels when from 0 to 5 gm. of sodium chloride are taken in addition to that contained in the food. We have tried various sites in the back for the implantations, but prefer the posterior axillary line. Six pellets or more may be inserted through a single small incision in radial fashion. Usually the pellets will last for six to nine months, sometimes being slowly and completely absorbed. Occasionally fibrosis about them seems to be so intense that absorption ceases.

Exertion in a patient with Addison's disease should be moderate, always kept short of fatigue or strain. Every effort should be made to avoid infections and even a mild coryza should receive careful attention. When active tuberculosis is present, the proper treatment of that disease should be carried on concomitantly with the therapy of the adrenal insufficiency.

## MAINTENANCE THERAPY

It is difficult to predict how long the intensive treatment of the crisis may be necessary. The nature of the treatment is determined by the response of the patient. Frequent blood pressure determinations are helpful. When the heart action is stronger, the pulse fuller, and the systolic blood pressure rises to 100 or over, the patient will usually be able to begin taking water, food and *salt tablets* by mouth. When food, salt and water intake by mouth are sufficient, intravenous therapy is stopped. Maintenance therapy is arrived at by gradually tapering off to it from the crisis treatment, and will vary considerably from patient to patient. If the patient tolerates large doses of salt, perhaps ten 1-gm. tablets daily in addition to 6 or 8 gm. taken in the food, comparatively small amounts of extract or of desoxycorticosterone are necessary. A few patients may be maintained on a high salt diet alone. Usually, however, it is best to give with the high salt intake a maintenance dose of extract or of desoxycorticosterone. If the large salt doses are unpalatable to the patient, part of the sodium may be supplied as sodium citrate, 5 gm. twice daily in orange juice.

If a high sodium intake with or without daily extract injections is employed, a *low potassium diet* such as that suggested by Wilder<sup>3</sup> is very helpful. Excessive potassium ingestion in such circumstances leads to prompt "decompensation" of the Addison's disease.

*Desoxycorticosterone acetate* supplies only part of the desired attributes of the adrenal cortex secretion, as we have discussed under the section on Pathological Physiology. It is particularly effective in causing retention of salt and water and in lowering the blood potassium. It has comparatively little effect upon the carbohydrate metabolism. Patients treated with desoxycorticosterone may have normal blood pressures, but may exhibit hypoglycemia with weakness, hunger and sweating. The maintenance dose of desoxycorticosterone acetate varies closely and inversely with the salt intake. From 1 to 10 mg. daily is the usual requirement. If 10 gm. of sodium chloride are taken daily, for example, only 2 mg. daily of desoxycorticosterone may be needed, but if the

## MANAGEMENT OF PSYCHOSES AND ALCOHOLISM IN GENERAL PRACTICE\*

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### THE PSYCHOSES

THE physician accustomed to dealing with affairs of life and limb will not readily appreciate the importance of a vague and variable symptomatology that rests on no demonstrable organic basis. But reflection upon the prevalence of functional nervous disorders and on the amount of disability and suffering they entail will begin to reveal their standing in medical practice. Dr. Logan Clendening in "The Human Body" tells of remarking to a distinguished colleague that "50 per cent of the patients whom I saw had no organic disease, but were sick because their minds, their souls, their lives were warped"; and the other replied, "80 per cent." (A British committee recently stated "something like 30 per cent" "in any group of sick persons.") A patient to whom Schroeder extended sympathy because of a recent acute illness, answered that she would rather have pneumonia all her life than nerves for one hour. Since the bulk of the psychoneurotic afflictions will inevitably fall to the lot of the general practitioner, the importance of his being able to understand and deal rationally with them is obvious.

### Diagnosis

That the making of a correct diagnosis is a necessary preliminary to successful treatment is in the case of the functional nervous afflictions more than a trite remark; it implies an obligation to gain a perspective view of an enormous mass of

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## BIBLIOGRAPHY

1. Kendall, E. C.: The Adrenal Cortex. *Arch. Path.*, 32: 474-501 (Sept.) 1941.
2. Hartman, F. A.: The Adrenal Hormones in Medical Practice. *J.A.M.A.*, 117: 1405-1408 (Oct.) 1941.
3. Wilder, R. M., Kendall, E. C., Snell, A. M., Kepler, E. J., Rynearson, E. H. and Adams, M.: The Intake of Potassium an Important Consideration in Addison's Disease. *Arch. Int. Med.*, 59: 367-393 (March) 1937.
4. Thorn, G. W., Howard, R. P., Emerson, K. and Firor, W. M.: Treatment of Addison's Disease with Pellets of Desoxycorticosterone Acetate Implanted Subcutaneously. *Bull. Johns Hopkins Hosp.*, 64: 339 (May) 1939.

in their own homes and their number is legion. They are still decidedly sick persons and when the physician is called to see them some questions that will arise in his mind are: What is a rational program of treatment for a nervous patient? What are the principles involved? What are the precautions to be taken?

In the attempts to give answers to these questions which follow, the method is adopted of considering the *principles* behind measures in common use, instead of describing cut and dried procedures themselves. In the home, procedures will have to be adapted to many extrinsic circumstances, and the necessary flexibility is best attained by devising on the spot measures that will put the desired principles into effect. As the writer has learned from experience in clinic and private practice, these principles can in some fashion be utilized for almost every patient.

It is again emphasized that the main bearing of prevailing methods of treatment is *psychological*. Bennett states that "this group of emotional disorders [*e. g.*, the minor psychoses] can be relieved only through adequate psychotherapy."

So the practitioner who wishes to deal with the psychiatric disorders will have to make the difficult step of abandoning his structural viewpoints for the moment and constituting himself an amateur psychologist.

### Principles of Treatment

Experience has shown that the most effective means of halting an acute psychotic episode or of breaking the back of a chronic, stubborn psychoneurotic state is to pluck the patient out of his familiar surroundings and plant him in a hospital bed for a stay of several weeks. What, then, are the principles effectuated by means of this maneuver? Analysis shows that they are chiefly (1) *rest*, mental and physical and (2) a *change* from a familiar to an unfamiliar environment.

*Rest.*—As just stated, the connotation of the term is mental as well as physical rest. The archetype of all methods of resting the psychoneurotic patient is the old Weir-Mitchell rest cure, which required the strictest enforcement of all details of the procedure; and *strictness*, if not of all the same details,

illness not yet clearly defined or understood. As far as present knowledge goes, even though they have been separated into types, the psychoses together belong to a generic whole, with an essentially similar etiology.

One type of diagnostic error is to consider the *chief complaint* in the "one-symptom neurosis" as a condition in and by itself. The terms "gastric neurosis," "nervous heart," "anorexia nervosa," "burning tongue" and (usually) "pruritus ani et vulvae" indicate misconceptions, for further investigation will reveal numerous, if less conspicuous, symptoms that will give the case its true mark. Similarly, such general subjects as "headache," "backache," "neuritis" and "arthritis" should not be studied without weighing the psychological element, since these have manifestations in common with neurotic illness. Whatever forms the symptoms may take—and they are innumerable—the underlying mechanism turns out to be broadly the same.

What, then, are the positive findings by which psychiatric illness is to be detected? Here as elsewhere, of course, snap diagnosis is risky, but a few lines of inquiry may quickly give the correct lead: (1) Ask about feelings of melancholy and despair with uncontrollable weeping. (2) Inquire into "nervousness," palpitation, smothering spells at night, nameless fears and excitability. (3) Investigate unusual insomnia, loss of appetite, feeling of exhaustion and pressure sensations over the vertex of the head. (4) Have there been, previously, nervous breakdowns? And find out what associates as well as the patient have noticed. After the suspicion of a psychosis is raised, inquiry on subsequent occasions may reveal the characteristic disproportion between the richness of the symptomatology and the paucity of organic findings.

### Emergency Aspects

There are, of course, different degrees of emergency. The more severe psychiatric cases call for immediate hospitalization; and in this relation it should be stated that for such patients every general hospital should have rooms set aside and suitably protected with inconspicuous steel screening.

The less disturbed patients are the ones who will be treated

affords a marked contrast to the home life; and so, also, to a lesser extent does staying at the home of a friend or going to another locality. Where a concession to necessity is made and a rest cure is carried out in the home, isolation is served by keeping the patient in one room and duplicating the hospital situation.

When an *attendant* or companion is needed, someone not of the patient's family is chosen. This is no reflection on anyone's character. The psychotic patient tends to "lean" on those close to him for moral support but he will gain self-confidence only by relying on himself. Many a therapeutic vacation to Florida or California has been in vain because a perfectly excellent spouse went along. The same applies even to daily walks, trips to the ball game or theater and other minor activities. If advisable, the patient should often go entirely alone.

But eventually the patient must return to his home life. Now isolation means *protection from anything that may excite or offend him*. The physician enters the home for a psychological survey and makes contact with each member of the family. Conflicts, clashing personalities, crudities, vulgarities, nagging and undue demands are searched for and, if found, corrected. Every member of the family is to understand that the sensitive nature of the patient cannot endure insults to ideals, pride and self-respect. Even discussions dealing with financial and personal worries are avoided in his presence. In a somewhat modified form this readjustment of the home life to spare the patient becomes a permanent schedule, arranged for the purpose of guarding against future trouble.

*Suggestion and Analysis.*—Popular books for the nervous patient are usually found to consist of finely woven, optimistic dialectics. This fact, as well as general observation, shows how important it is that encouragement by word and act should deliberately be contrived to sustain the hopes of the patient, whose fear and despair are beyond anything the well person can conceive.

When he is in a more objective frame of mind, the patient can profitably *analyze* his own conflicts, complexes and mental tendencies. He learns that pains and sensations of all sorts

is still an essential element of rest cures. The patient is kept almost continuously in bed for from three to six weeks. Mental rest is enforced by denying the patient visitors, reading matter, the radio, telephone communication and all but necessary conversation with attendants. (Naturally, after a week or two selected exceptions are introduced.) These exacting requirements are necessary, a psychosis being what it is.

When treatment is carried out *in the home*, this hospital situation is duplicated almost precisely. A room is set aside, the patient remains in bed, external sounds are excluded, meals are served on a tray, and only those enter who are engaged in care. Clearly a nurse will greatly facilitate the designs; otherwise a suitably objective member of the family renders the necessary attention. A similarly complete seclusion will always be provided whether the patient is to rest for a week, a week-end or a few hours.

The physical rest is necessary because evidently the constitution of the psychotic lacks its full quota of endurance and reserve. *This fact must always be kept in mind.* Further, the presence of a psychosis is almost invariably attended by a state of exhaustion that requires some time for its recuperation.

*Isolation.*—This principle is of the very essence of psychology. It is the force that chiefly gives value to our vacations and, as well, the one that is largely responsible for the benefits derived by the nervous patient from hospitals and sanatoria. Merely breaking off old associations and changing to an unfamiliar environment has the effect of destroying unwholesome trains of thought and emotional reactions and of allowing more normal viewpoints to become established. The term, "isolation" is used in the sense of separating the patient from associations that are bad for him whether in themselves or by virtue of habit. In practice it means slightly different things at different stages of the illness.

First, when the psychosis is in the acute phase, isolation means *separation from all familiar contacts*—home, family and work—and finding surroundings in all respects as contrasting with the usual ones as possible. This overt move is necessary because the maximal effect is desired. The hospital obviously

doses three or four times a day may be indicated for a few days; and thereafter as necessary for periods of unusual nervousness. The chief reliances for sedation, however, will be the old favorite, sodium (or triple) bromide (about 15 grains, three or four times a day) or elixir alurate or elixir ipral (in teaspoonful doses three or four times a day, either as they come or as, very often, diluted with equal parts of water). Elixir nembutal is slightly stronger and will usually have to be diluted with water.

The writer has little faith in the usual *tonics* and some, such as strychnine and thyroid, are positively harmful. The *bitter stomachics* may be helpful in promoting appetite; daily injections of small amounts of *insulin* (say 20 units) have also been employed for this purpose.

The *exhaustion* so often complained of, however, is a tough problem. It is probably more a neurotic sensation than a real state. The sedatives, curiously enough, may be of service. Benzedrine sulfate (5-mg. doses, three times a day) has been recommended. It is sometimes very effective but should be utilized cautiously as the effects may be undesirable; at best it is probably of service only in occasional and milder cases. The writer has had some satisfaction from the proprietary, aminoids (Arlington Co., a dessertspoonful three times a day) said to be a preparation of amino acids. The old remedy, sodium cacodylate (7 grains), given into the muscles of the hip twice a week for a few weeks may have a favorable result.

The *endocrine and vitamin preparations* are still in the controversial field, although they seem to be widely used by practitioners on assumptions that are not always well defined. Psychiatrists still do not manifest in their writings any great enthusiasm for them, although some speak of the possible value of a general tonic effect. For this purpose theelin in oil may be given in daily injections of 1 mg. (10,000 units) for a couple of weeks and thereafter twice a week for a few weeks longer. The new stilbestrol (Squibb) is much cheaper but should be used with some caution. Among the vitamins, the favorites are those of the B complex, specifically thiamine hydrochloride and nicotinic acid. Thiamine hydrochloride can be tried in amounts of 10 to 25 mg. hypodermically three

can have a mental origin and do not indicate, as he is apt to believe, a dreadful disease. He comes to realize that conflicts between passions and conscience are the lot of mankind. His prejudices, phobias, hates and desires are analyzed into their component factors of self-defense and emotional over-reactions. The value of such analysis is, of course, greatly stressed by certain schools of psychiatric thought; and the good results of even superficial self-examination undoubtedly justifies its employment. The physician therefore should spare the time for numerous *confidential interviews* with the patient extending often over a period of months.

In reviewing the foregoing outline, the program of treatment is found to run through the stages of (1) stringent measures to halt the psychosis, (2) convalescent measures, bridging the return to normal activities and (3) a regulated life, to take account of the patient's mental and physical limitations. Measures for carrying out these various purposes include such expedients as a rest cure in the home, resting at the home of a friend (or even boarding out, for the isolation), vacations to the country or in another climate, hunting and fishing expeditions of various lengths from days to weeks, various degrees of isolation in the home and a schedule of daily life. (Occupational therapy, consisting of handicrafts and including carpentry and gardening, is an unexcelled diversion for convalescence and a hobby for the future.) The measures will have to be fitted to the illness, the financial circumstances and the family situation, and much ingenuity and study will need to be exercised by the physician. The management of the psychotic patient is clearly no simple and easy undertaking.

### Medication

It is unthinkable under the present conditions of medical practice that the nervous patient will be treated without resort to *sedative preparations*. These are valuable not only for the necessary purpose of securing adequate sleep but also for holding in check nervous impulses and restlessness, thereby permitting a measure of self-control to become established.

For the very restless patient, sodium amytal in 3-grain

The *treatment* is as previously outlined, without special features. But the real opportunity comes in the intervals, when proper care can prevent further trouble. The patient's home life, the nature and the amount of the work he does and his diversions should be strictly regulated. It is well for him to see the physician concerning these matters at frequent intervals whether there are any special complaints or not.

*Dementia Praecox.*—Here intellectual processes themselves are involved, delusions and hallucinations being common though not invariable symptoms. The patient may merely be seclusive, inattentive and disinclined to effort. Surges of anger or other irresponsible reactions without adequate incentive occur. Associates notice a change in character; the patient impresses them as being another person. The degree of behavior disturbance varies from case to case.

In this psychosis the physician has a chronic, pernicious disturbance to deal with and the most stringent and prolonged care will be called for. The patient can rarely be treated entirely in his own home. If he does not go to a hospital, he should have a palliative rest cure at home with isolation in all its implications rigidly enforced; then he should have a change of scene, on a farm or in the home of a relative. There he at first entertains himself, under supervision, and later may take up some light occupation which seems to please him. Owing to the chronic nature of these cases care will have to continue for months, and one should not be misled by seeming remissions. Back in his home, the patient should be interfered with as little as possible, contacts on personal matters being limited chiefly to the physician.

A permanent occupation for these patients ought to be generally wholesome and not too exacting. An academic or clerical career is rarely suitable. Keeping a shop, working in a filling station, or factory work is preferable. Marriage is inadvisable.

*Involutional Melancholia or Agitated Depression.*—This type of psychosis comes on in the involutional period of the sexual life and, surprisingly, its onset sometimes coincides almost exactly with the beginning of menopause. But men also are affected. The clinical picture is a mixture of the hyper-

times a week and nicotinic acid, say 50 mg. two or three times a week for a few weeks. Allergic reactions occasionally occur with the vitamins; but the writer still favors the hypodermic route, for one reason, because it keeps the physician in touch with the patient. Moersch says, "If there is no prompt response to the [vitamin] treatment it should be discontinued. If improvement does occur, the possibility of psychotherapy playing a role in this improvement must be weighed carefully."

During a psychosis and the period of convalescence, *soporifics* are often indicated and their use is not objectionable so long as other measures of treatment are continuing. Sodium amytal (3 grains) seconal ( $1\frac{1}{2}$  grains) and nembutal ( $1\frac{1}{2}$  grains) are commonly used but should not be repeated more than once in a single night. Sometimes the sedative is not taken at bedtime but during a wakeful period later, thus avoiding a second dose.

### Metrazol Shock Therapy

This amazing therapeutic measure, which is one of those totally unexpected boons to which medicine is occasionally treated, is mentioned only for the sake of completeness, for it will likely always have to be administered by the expert. Never to be followed in a stereotyped fashion, when properly individualized, metrazol therapy is capable of achieving results that in comparison with former standards amount to the miraculous. It is utilized in all types and degrees of functional psychotic involvement; and there is promise that it will even invade the field of the neuroses where these are severe and long-continued. The rationale of metrazol therapy is still quite obscure but its usefulness seems to be firmly established by practical results.

### Special Features of the Different Psychoses

*Manic-depressive Psychosis.*—Manic and melancholic phases or disturbances of mood alternate in this type. The manic patient is hyperactive and elated while the depressed patient is hypo-active and despairing. In the intervals between attacks, patients are neurotic and high-strung.

Extremely prevalent are the instances where the chief complaints are headache, feelings of exhaustion, "neuritis," "arthritis," "sinusitis" and the like. The "conversion" of the mental conflict into symptoms suggestive of physical disorders is a well known phenomenon. Only special inquiry into the questions of insomnia, nervousness, fits of depressions and of weeping and so on reveals the true nature of the case. These cases by all means are to be treated in the same fashion as other psychotic cases. *It is a great mistake to put much stress on the chief complaint*, for to do so only "fixes" the symptom psychologically, more often than not with a bad effect. On the other hand, as soon as general measures are instituted the presenting symptoms automatically begin to fade away.

#### ACUTE ALCOHOLISM

The sufferer from acute alcoholism usually is either a dipsomaniac who has been on a prolonged debauch or a chronic drinker whose condition has reached a crisis. The immediate treatment is much the same in the two instances; but in the latter case treatment will have to be continued longer and the complication of an alcoholic psychosis is liable to occur. Delirium tremens and the "wet brain" as signalized by prolonged stupor, labored breathing and cyanosis are distinctly conditions for hospital care.

The essentials of the treatment of an acute alcoholic state are the *withdrawal of the alcohol* and keeping the patient quiet through different degrees of mental disturbance and rebelliousness. A good rule for the withdrawal of alcohol is decreasing the amount 50 per cent daily. If the alcohol is diluted with water and the total amount of the dose always kept constant, the patient will not know how much alcohol he is receiving. In the home particularly, the psychological factor will loom large and a nurse will greatly facilitate progress.

The mental irritability is to be taken care of by means of *sedatives* in large amounts. Sodium amytal seems preferable to the old favorite, paraldehyde; but large doses are to be avoided while there is much alcohol in the stomach. The writer often orders 3 grains of sodium amytal every two hours until the patient becomes drowsy, and thereafter less fre-

activity of the manic and the depressed mood of the melancholic. In milder cases the patients are "nervous," anxious, exhausted and have "hot flashes." The course varies widely according to the degree of the involvement, severe cases being stubborn and milder cases improving quickly, but tending to recur.

As to therapy, the age of onset of this class has lent great plausibility to the idea of an endocrine disturbance. Indeed "menopausal psychosis" is widely used, by nonspecialists, for all psychoses arising in women at this age period. But ample experiments under controlled conditions have not borne out this view. A number of investigators have treated groups of patients with large amounts of estrogenic substances and have continued observation for months; and as a result have detected only an insignificant influence. Furthermore, it is to be noted that these involutinal cases, whether severe and long-continued or mild, are among the most promising for metrazol shock therapy. Hence we can but conclude that this endocrine factor is unestablished.

There remains, however, a vast field of disorders, many of a milder degree and treated by practitioners in their offices, of which "nervousness," hot flashes and "vasomotor disturbances" are symptoms, which remain unclassified as between psychoneurosis and "menopausal disturbances." These conditions shade without a break into the psychoses and no point can be detected where one etiologic factor ceases to act and another begins. These patients are now commonly treated with *theelin*, sometimes in huge amounts (24,000 units daily for two weeks); but their real classification would appear to remain one of medicine's major problems.

There has been of late less tendency to stress the making of dogmatic diagnoses in the case of the psychoses because of the number of intermediate cases that partake of the character of more than one type. The present inclination is to try to judge of severity of involvement from the depth of disturbance or the course, rather than to reach conclusions from a few symptoms; and to proceed on that basis.

Further it is to be recognized that *not all* cases of psychoses by any means announce themselves by psychic symptoms.

residence may avoid certain temptations. The most hopeful expedient in prospect is the national movement known as *Alcoholics Anonymous*, in which former alcoholics get together periodically and lend each other moral support. In almost any community the physician can find enough subjects to install a chapter of this beneficent movement.

quently. Often eight doses of this amount are given in twenty-four hours. When it cannot be given by mouth, special rectal capsules are available or the ordinary capsule may be given by rectum.

When the patient is very disturbed, *wet packs* are very useful. A sheet is wrung out of cold water and wrapped tightly about the patient whose arms are fastened tightly against his sides. Then snugly over the sheet a heavy blanket is wrapped and pinned tightly with large safety pins from over the shoulders to beneath the feet. The patient is kept in this pack for three or four hours, the pulse and color meanwhile being watched.

*Complications* may arise, although they are not very common. With the advent of sulfonamide drugs, pneumonia is not the tragedy it once was, even in older persons. If the patient is greatly dehydrated from vomiting, he will have to get fluids somehow. Retention of urine may occur. Where there is much vomiting, sedation may have to be administered hypodermically, the H.M.C. (hyoscine, morphine and cacaine) No. 1 or No. 2 tablets being an effective combination.

In the chronic alcoholic, the acute condition may be succeeded by a psychosis, characterized by a failure of memory, a condition which may continue over weeks and still eventuate in recovery. No special treatment beyond nourishment, hygiene and soporifics is called for.

*Thiannine hydrochloride* is recommended for the alcoholic, according to Jolliffe, in amounts of 20 to 50 mg. daily for three to six weeks and less frequently afterwards. The diet should be full and well balanced, not only during treatment but also thereafter for its prophylactic value.

The large problem in the case of the alcoholic patient is the *prevention of a recurrence*. There seems to be no power within human control that will prevent the patient, so inclined, from returning to the use of alcohol. The usual appeals and arguments sooner or later fail in their purpose in a large proportion of instances. Physical and mental health should be supported by all means known to the ordinary physician and the psychiatrist. Overwork and undue emotional stresses are spared the patient as far as possible. A change of occupation or

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## EMERGENCIES ASSOCIATED WITH RESPIRATORY DISEASES\*

R. O. MUETHER, M.D., M.S. (Int. Med.)†

### PULMONARY THROMBOSIS AND EMBOLISM

ONE of the most dramatic happenings in medicine is the collapse of the patient with a pulmonary embolus or thrombus. This condition, which is seen both by the internist and the surgeon, is in the truest sense an emergency. It is unfortunate but apparently true that it is increasing in frequency. Why this should be true is a matter of debate. It might be pointed out that anesthesia, increase in age of patient and prolonged rest in bed seem to play a role in the incidence of thrombi and emboli generally and particularly in the patient with vascular disease.

The etiology of the condition is obscure but it is known that *damage to the vascular tree* is a predisposing cause and, therefore, any condition which produces toxins of such nature as to injure the intima of the vessels, or which places an unusual strain on already damaged vessels, may lead to the development of thrombi or emboli. It is difficult to believe, as some apparently do, that the vessels of the lesser circulation are not injured and damaged by the same conditions as the vessels of greater circulation. It is more likely that the greater circulation is simply studied more carefully since it is more available. Careful dissection of the vessels of the pulmonary circulation would no doubt tend to change our concept of lesser circulation disease. There is little doubt in my mind that not a few deaths which occur with respiratory symptoms and in respiratory disease

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the development of *shock* as evidenced by diminished blood volume and hemoconcentration; if the shock is sufficiently severe, there may be a fall in the blood pressure. The patient's symptoms may grow worse after the onset and cyanosis may develop; later frothy sputum containing blood may be expectorated and, in rare cases, frank hemorrhage may occur.

Physical examination shortly after the accident may reveal very little; a friction rub may be heard at any time from a few hours to several days; evidence of consolidation may become noticeable shortly after the onset of the condition. The lesion usually shows evidence of resolution in a few days unless secondary infection develops—then it will run the course of a nonspecific pneumonia.

*Occlusion of Small Vessels.*—There can be little doubt that obstructions of the small vessels of the lung can occur and that such emboli lead to very little trouble. There may be a *sudden pain* in the chest associated with a very mild degree of *shock* characterized by a feeling of faintness or dizziness, the pulse rate may be but slightly altered, a moderate tachycardia may be present. The temperature of the patient may actually be subnormal at first, to be followed in an hour or two by a slight elevation. It is unusual to find any change on physical examination. Careful examination may reveal increased breath sounds and occasionally increased voice sounds as well. The typical findings of consolidation are not present and the roentgenogram will almost always fail to show evidence of a pathologic process.

*THERAPY.*—It is obvious at once that the primary consideration in the treatment of these conditions is the treatment of the *shock* that accompanies them: in thrombosis or embolism of the large vessels, death follows so quickly that therapy cannot be instituted. When the very small vessels are involved, treatment is unnecessary or at most resolves itself into bed rest and symptomatic treatment for pain. It is in the involvement of the medium-sized vessels that therapy offers most to the patient.

Since prevention is always the most important part of therapy, it is perhaps well to point out that *careful attention to the circulation* of the patient both before and after surgery,

are the result of pulmonary thrombosis and not, as they are so frequently recorded, heart failure.

The symptomatology and prognosis of pulmonary infarcts depend upon a number of factors, not the least of which is the rapidity and the location of the occlusion: the more rapid the occlusion and the larger the vessel involved, the more severe will be the symptoms and the graver the prognosis. We can then classify this condition into thrombi of the large, medium and small vessels, each class being modified by the rapidity with which the thrombosis takes place.

**SYMPTOMS AND SIGNS.—Occlusion of Large Vessels.**—The sudden and complete occlusion of a main pulmonary artery leads to marked *dyspnea*, striking *cyanosis* and oftentimes *sharp, severe pain*. The patients go into deep shock and usually die before much in the way of therapy can be attempted.

The incomplete occlusion of a large pulmonary vessel may go unnoticed until the vessel is more completely occluded or the patient may complain of feeling uncomfortable—he may be *restless*, slightly *cyanotic* and complain of a *cough* which may be dry or may result in the production of a frothy, bloody sputum. Roentgenograms are usually not very helpful at this time and therapy consists primarily of sedation and bed rest. Almost all of these cases go on to complete occlusion and the end-result will be determined primarily by the size of the vessel occluded. Occlusion of the large vessels invariably leads to death.

*Emboli* of the large vessels result in symptoms which are indistinguishable from those of thrombi and are almost always of sufficient size to obstruct the vessel totally, thus leading to very sudden death.

**Occlusion of Medium-sized Vessels.**—Thrombosis or embolism of the medium-sized vessels is accompanied by signs and symptoms similar to but less severe than those of large vessels. Usually there is a sharp *pain in the chest* not unlike that seen in pleurisy and, in some cases, a *pleural rub* can be heard. The patient usually has a feeling of *apprehension*, the respiration is shallow and slow and there may be a limitation of motion of the chest on the affected side. The pulse rate is increased and the temperature may be elevated—all this is combined with

Pulmonary hemorrhage may follow or be associated with either acute or chronic disease. Pneumonia, tuberculosis and whooping cough are the acute respiratory diseases most often complicated by hemorrhage while lung abscess, bronchiectasis, gangrene of the lung and spirochetal bronchitis are the most common chronic pulmonary diseases complicated by severe bleeding. Parasitic infestations of the lungs may cause hemorrhage but these are rare. Neoplastic growths of the respiratory system and mediastinum may also cause bleeding.

Blood dyscrasias, cardiovascular disease and trauma account for most of the extrapulmonary diseases which can produce pulmonary hemorrhage.

It is well known that bleeding is one of the commonest symptoms of pulmonary disease but it is equally evident that severe hemorrhage is relatively uncommon and this is explained most easily by assuming that in most respiratory diseases the bleeding, which occurs, is the result of increased intracapillary pressure and permeability without the presence of a great deal of necrosis or ulceration; when ulceration does take place, as it does in tuberculosis and lung abscesses, there is usually so much thrombosis in the small vessels that they are no longer capable of bleeding. Severe hemorrhage, then, occurs as an accident to an already damaged lung in which the vessels have not been occluded by thrombus formation. Sudden strain, such as lifting, coughing or a blow, may increase the pressure suddenly within a damaged vessel and thus start a severe hemorrhage. It is frequently stated that pulmonary hemorrhage is never fatal. This is not true and every effort toward stopping excessive bleeding from the lung should be made as quickly as possible.

**DIAGNOSIS.**—The diagnosis of pulmonary hemorrhage is, of course, obvious when the patient expectorates a large amount of blood. If the bleeding is begun at the patient's home, he is prone to exaggerate the amount of bleeding which has taken place but the patient's story should not be taken too lightly since his history may provide quicker information on the severity of the condition than anything else.

The diagnostic problem, which must be faced, is the de-

as well as during respiratory infections, may do much to prevent and reduce the incidence of these conditions. The patient should be given sufficient cardiac stimulation to maintain adequate circulation if this is indicated. The position of the bedridden patient should be changed from time to time and excessive sedation should be avoided.

Every effort should be made to *avoid dehydration* and, for this purpose, some criterion other than the condition of the tongue and the elasticity of the skin should be used. The hematocrit may be used and an effort made to prevent an increase in the cell volume. If it is necessary to administer an anesthetic, it should be remembered that this procedure has a tendency to increase the proteins and the platelets and thus increase the chances for pulmonary emboli and thrombi infection. Excessive manipulation at operation will also increase the likelihood of pulmonary emboli.

Actual therapy for embolism is very limited. In embolism of the large vessels *embolectomy* may be performed if the patient survives the initial shock of the disease. Embolectomy is a hazardous procedure and requires a skilled team for its successful performance. The best results are obtained when a special group is organized as an embolectomy team. Such a group then practices at frequent intervals on animals or cadavers, so that they become letter perfect in their various functions.

The treatment of shock will be discussed later and except for this, treatment is symptomatic. *Heparin* given by continuous drip may prove of considerable value in the slowly occluding vessels if the diagnosis can be made. I have had no experience with this material for this purpose but it should prove invaluable when embolectomy is done.

#### PULMONARY HEMORRHAGE

Equally as alarming as pulmonary thrombosis or embolism, both to the patient and the family of the patient, is pulmonary hemorrhage. Hemoptysis occurs even more commonly in actual disease of the respiratory tract than do embolism and thrombosis but, like these, it may also occur as a result of disease remote from the respiratory tract.

## ATELECTASIS

Partial atelectasis is not usually of such severity as to result in an actual emergency but massive collapse of the lung very definitely is a medical emergency. It is true that the initiating factor in massive collapse of the lung is still uncertain and three theories have been proposed: (1) the *mechanical* or obstructive; (2) the *infectious* in which it is suspected that pleural and diaphragmatic inflammation leads to physiologic changes which produce atelectasis; (3) the *nervous* in which impulses from the nervous system are thought to produce the collapse. It is entirely possible that all three play a role and that the importance of each one varies with the particular case.

**SYMPTOMS AND SIGNS.**—The onset in massive atelectasis is sudden and there is marked *dyspnea* and *cyanosis*; pain is not as a rule severe and there is very little cough. *Fever* may be very high, have a remarkably rapid onset and is usually greater than that expected with pneumonia; *tachycardia* and *rapid shallow breathing* are also outstanding symptoms in this condition. The patient usually insists upon sitting up and his appearance suggests impending death. In this condition, as in embolism, *apprehension* on the part of the patient is usually great.

Examination of the patient reveals absent or shallow respiration on the affected side; the heart is drawn toward the atelectatic side as evidenced by the location of the apex beat, dullness to percussion being associated with an absence of breath sounds over the involved side. The condition pursues a variable course; if it is due to mechanical obstruction, the removal by bronchoscope or by coughing of the obstructing agent will allow the condition to clear almost at once; if this does not occur it may persist for several weeks.

**THERAPY.**—The treatment consists of treating the patient for shock and attempting to expand the lung. In some instances, depending on the etiology, the obstruction can be removed *bronchoscopically*; or, if this is not feasible, *oxygen* and *carbon dioxide* may be administered either by nasal catheter or face mask to increase respiration and expansion.

termination of the cause of the bleeding. In many instances this is not difficult, since it may be known that the patient is suffering from one or another of the pulmonary diseases which lead to such an emergency. If there is no known history of disease of the lung, the patient should be examined carefully but thoroughly and x-ray pictures taken if necessary. It is often difficult to determine the point of bleeding when the patient has bilateral disease but *increase in the rales*, occasional *pain* and discomfort, as well as *changes in resonance* on the bleeding side are helpful.

THERAPY.—The patient must be kept at *absolute bed rest* and anxiety should be allayed with sedatives. *Morphine* in full doses is perhaps the most satisfactory, although *codeine* is sometimes preferred. The physician should use the drug in a sufficient dose to produce rest and relaxation of the patient and for this purpose large amounts may at times be required. *Vitamin K* may be administered if the prothrombin time is prolonged. *Calcium* is often administered though very little is accomplished by it. *Moccasin venom* administered subcutaneously will decrease the clotting time and may be useful. There is no contraindication to *blood transfusion*, particularly if the erythrocyte count or hemoglobin determination has shown a marked fall.

Patients who have been bleeding excessively may show signs of shock or collapse and appropriate therapy must then be instituted.

In severe hemorrhage an attempt may be made to *collapse* the lung from which the blood is coming. Such a procedure is not particularly dangerous. When it is attempted, the lung should be collapsed as completely as possible, with fluoroscopic confirmation if at all possible. Once the lung has been collapsed, it should not be allowed to re-expand for some time.

The difficulties which arise in attempting to collapse a bleeding lung are associated primarily with the difficulty of collapse due to pleural adhesions, difficulty in determining the side from which the blood is coming and the danger of peripheral vascular collapse or shock.

The prognosis in these cases is very poor, particularly if the patient has been a previous sufferer from nephritis or cardiac disease. Attempts to correct the underlying pathologic process, treatment of the shock and provision insofar as possible of relief of symptoms make up the treatment. In those cases in which cyanosis is a factor the use of *oxygen* and *carbon dioxide*, or *oxygen* and *helium* is indicated. It is the consensus that acute pulmonary edema is the result of stasis in and increased permeability of the lung capillaries and, therefore, measures which tend to increase the circulation through the lungs and, at the same time, diminish permeability of capillaries are indicated. Such measures are to a considerable extent identical with those used in the treatment of shock.

#### PNEUMOTHORAX

Pneumothorax develops most frequently in tuberculosis, which accounts for at least 70 per cent of spontaneous cases. The remainder may occur in healthy individuals or as a complication of gangrene of the lungs, abscess, bronchiectasis, hemorrhagic infarct and rupture of an emphysematous blister or bleb. Traumatic wounds which perforate the chest wall may also produce a pneumothorax which is usually complicated by the presence of blood, forming a hemopneumothorax. It should be noted that the incidence of this condition is no doubt reduced by the fact that pleural adhesions of any degree will prevent the collapse of the lung even if there is a small perforation or tear in the lung parenchyma.

**SYMPTOMS AND SIGNS.**—The onset of this condition in the majority of patients is very sudden and leads to the characteristic symptoms of *sharp, agonizing pain, peripheral vascular collapse, dyspnea* and, in some instances, *cyanosis*. The pain is rather typical, being tearing in nature and very severe. The anxiety, weak pulse, fall in blood pressure and increased perspiration with paleness all point to *shock* as a most important consideration.

*Physical examination* reveals, in addition to those points already mentioned, absence of respiratory movements on the affected side with a loss of Litten's shadow on the same side. Tactile fremitus on the affected side will be absent or mark-

Rolling the patient from side to side, light pounding over the affected side and forceful expiration may aid the patient.

In some cases the anxiety of the patient may be so great that small doses of *morphine* are indicated. If the condition persists, infection may occur in the atelectatic areas and therapy must then be directed to this condition.

#### PULMONARY EDEMA AND CONGESTIONS

Acute pulmonary edema is an important complication which may follow any severe irritative lesion in the lungs and may also be associated with acute myocardial failure. It is not unusual to have the patient develop massive edema of the lung in any of the severe respiratory diseases like pneumonia, abscess of the lung and acute pulmonary tuberculosis. The various pulmonary irritants, particularly those used in chemical warfare, give rise to their deleterious effects by producing edema of the lungs. In civilian life ammonia and other refrigerants may, as a result of accidental inhalation, lead to severe pulmonary irritation and edema.

Pulmonary congestion may also result from lung irritation due either to infection, toxic gases or cardiac failure. The clinical and other observations are closely similar to those in pulmonary edema; in fact, the differentiation is one of degree rather than kind.

**SYMPTOMS AND TREATMENT.**—The attack usually begins with startling abruptness and, in the case of pulmonary irritants like chlorine, phosgene and the fumes of nitrous and nitric acid, it may have its onset twelve to twenty-four hours after the patient has been removed from contact with the irritating gas. In these cases *dyspnea* may be marked, *cyanosis* is noted and coughing is at a minimum. The *sputum* may be thick and streaked with blood. The patient with massive pulmonary edema is extremely dyspneic, and brings up as a result of an irritating cough a great deal of frothy, bloody sputum. The physical examination yields positive data only on auscultation, as a rule. The lungs are filled with rales, the breath sounds are diminished, and the heart sounds are heard with difficulty. In advanced cases, dullness and coarse fremitus may be noted by palpation and percussion.

information with considerable clarity. The use of diagnostic methods should, however, be emphasized. The presence of *hemoconcentration* in shock is admitted by all and this fact provides a definite approach to diagnosis and prognosis. The red blood cell count, the total proteins, the hematocrit reading, as well as the specific gravity of whole blood or plasma, all serve as rather delicate indices of hemoconcentration. It is, of course, essential that the determinations be made before the onset of shock in order to evaluate the later results properly; however, in the case of the specific gravity and hematocrit the later changes are usually of such a magnitude as to be helpful whether they had been previously determined or not. The technic for all of the tests with the exception of the specific gravity test is well known and need not be enlarged upon.

*Specific Gravity Test.*—The introduction of the *falling-drop method* for determining specific gravity and from the specific gravity the *total proteins* after the technic of Kagan and others has made this method very practical for the study of shock; the determinations can be done on a small amount of blood; if whole blood is used a finger stick is all that is necessary. The method consists, in brief, in timing the fall of a measured drop of blood, plasma or serum through a measured distance in a medium of known specific gravity and temperature. Having secured the time it takes the measured drop of material to fall the measured distance, the specific gravity is determined by referring to a suitable chart. The entire procedure can be carried out in the space of two or three minutes and the process may be repeated as frequently as necessary.

*TREATMENT.—Rationale.*—Having established the fact that the patient has hemoconcentration, the therapeutic endeavors should lie in the direction most likely to produce dilution without at the same time lowering the osmotic pressure too much. It must be kept in mind then that in shock the capillaries are injured so that they are usually dilated and have increased permeability. The mechanism responsible for these changes is not well understood. Nervous mechanisms have been postulated along with theories which involve the liberation of toxins such as the "H" substances and the alterations of

edly diminished and the heart as indicated by the apex beat will be displaced toward the sound side. The use of percussion reveals increased resonance on the affected side which varies from a slight increase to a definite tympanitic or drumlike sound, depending on the amount of pressure which has developed on the involved side. Percussion will also confirm the shift of the heart and the mediastinum to the sound side. When the stethoscope is applied to the involved side it is usual to find a complete absence of breath sound but on occasion the breath sound may be of normal intensity or even increased. Voice sounds cannot as a rule be heard. The metallic tingle, the succussion splash and the coin sound should be sought for but unfortunately they are often absent. When these sounds are present they are pathognomonic of pneumothorax.

**TREATMENT.**—The treatment consists primarily of *treating the shock* and using *supportive measures* such as heat, oxygen and, when the pain or apprehension is great, morphine in full dosage. In many cases the increased pressure may be so great that the heart and opposite or unaffected lung may be rendered ineffective; in such cases the pressure within the pleural cavity of the involved side should be reduced by *aspiration* of some of the contained air—only a sufficient amount should be removed, however, to relieve the acute symptoms.

### SHOCK

The emergencies which have been discussed are but a few that the physician may be called upon to face during the course of acute or chronic pulmonary disease. The conditions mentioned and, for that matter, nearly all pulmonary emergencies become formidable in proportion to the degree of peripheral vascular collapse which they produce. Peripheral vascular collapse is similar if not identical to so-called surgical shock and the physician must bring into play the same diagnostic and therapeutic methods which have been used so successfully by the surgeon.

**DIAGNOSIS.**—It would profit us but little to enter into a discussion of the etiology of shock since recent monographs by Scudder and Moon and others have presented the available

careful observation of the patient; at the first sign of returning shock, the treatment should be repeated. Best results are obtained in my experience when both suprarenal cortical extract and plasma are used.

*Heat.*—The use of heat is also considered very important in handling the patient in collapse. Blankets and warm water bottles still stand at the fore for this purpose; if available, however, the electric cabinet or the electric light body cradle is very helpful. Recent investigation by Wiggers and others tend to show that shock develops more slowly in animals which are cooled than in other animals which are kept warm. Just what influence these observations will have on our methods for the treatment of shock cannot be stated as yet. It seems to me to be of such significance that we should keep ourselves alert for their possible practical application.

*Oxygen.*—Oxygen or oxygen and helium given by face mask, catheter or tent, if administered continually in sufficient amounts, will aid materially in the patient's struggle against shock. This is particularly true in respiratory conditions, since in many cases the blood is inadequately oxygenated due to the primary pathologic process in the lung.

#### SUMMARY AND CONCLUSIONS

Medical emergencies which arise as a result of respiratory diseases have been discussed in brief. It is not intended that this discussion be considered complete as there are many other emergencies which may arise during the course and in the convalescent period of respiratory disease. It has been my purpose to indicate the close similarity, insofar as altered physiology is concerned, in many of the emergencies which do arise. It seems to me to be helpful to think of many of the changes which take place and the symptoms which arise as resulting from peripheral vascular collapse or shock. If this concept is accepted the logical therapy for the general symptoms immediately suggests itself.

Methods for the treatment of shock along with therapy for the more local manifestations of the emergencies have been outlined.

electrolytes. Scudder has emphasized the changes that take place in the electrolytes, pointing out that in shock there is always an increase in the serum potassium and a decrease in the serum sodium. Once the mechanism of peripheral vascular collapse is initiated, other factors may soon play a role; the volume output from the heart is diminished primarily because the blood is trapped in the periphery, the tissues soon suffer from oxygen want and this tends to aggravate the damages which have already affected the tissues including the capillaries. There is a continued loss of fluids and some protein into the tissues. If relief of these conditions is too long delayed the process becomes irreversible and the patient dies.

*Fluids.*—The treatment of shock in the various conditions mentioned consists, then, of stopping or correcting insofar as possible the condition which has produced the shock, while at the same time an attempt is made to overcome the dangerous effects of peripheral vascular failure. Since dilution is unquestionably needed, additional fluids should be supplied and these should be of such a nature that they will not be lost rapidly from the blood stream. *Plasma* is the most satisfactory solution to administer in this condition, since it can be kept readily available and can be given without typing or cross-matching. It should be given in fairly large amounts and as soon as possible. The use of a liter or two is not excessive and it may be given repeatedly as indicated, the hematocrit or specific gravity serving as a guide for the progress which is being made. *Acacia*, and more recently *pectin*, have been proposed as substitutes for plasma but it is still doubtful whether these substances are as satisfactory as they might be. In those instances in which excessive loss of blood has occurred, it is wise to resort to *whole blood* as the restorative although plasma may be administered while the blood is being prepared for administration.

*Suprarenal Cortical Extract.*—The use of suprarenal cortical hormone in rather large doses is also very helpful in the treatment of shock. It is wise to give 10 to 20 cc. of the extract along with intravenous injections of a solution of sodium chloride; if the patient shows a good response, as indicated by a fall in specific gravity, further treatment may be based on

## MANAGEMENT OF EMERGENCIES DUE TO ALLERGY\*

KEITH S. WILSON, M.D.†

THE hypersensitive person is notorious for the sudden explosive violence of his reactions. He is peculiar in that often the stimulus that precipitates the crisis is a simple substance, harmless to the majority of individuals.

### ACUTE BRONCHIAL ASTHMA

At the height of an attack of asthma, there is an outpouring of tenacious mucus, edema of the mucous membrane and spasm of the musculature of the bronchi. This causes such obstruction to the air passages that intense dyspnea results. All these effects are the consequence of increased vagus stimulation. The following measures are designed to combat the underlying process.

**SYMPTOMATIC TREATMENT.**—1. *Epinephrine* (Adrenalin).—Of all the drugs used in allergy, epinephrine is the most generally useful for all the various allergic crises. It is a life-saver in the control of acute symptoms, often warding off death. Its effectiveness resides in the stimulation of the sympathetic nerve endings which produce results antagonistic to the parasympathetic (vagus) or cholinergic fibers. It thus combats edema and smooth muscle spasm.

The recommended dose of 0.2 to 0.3 cc. of the 1:1000 solution is given subcutaneously and provides relief in a few minutes in uncomplicated cases. These amounts are small and may be repeated several times at hourly intervals. Infants and children tolerate such doses. Injections of 0.5 to 1.0 cc. are usually too large and are prone to cause side-reactions such

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given at onset of a paroxysm when the mucus is very tenacious and should be continued until the sputum is fluid. A convenient prescription is:

Potassium iodide .....	10.0
Elixir of lactopep .....	120.0
Sig.: One teaspoonful every three hours.	

Another favorite prescription is:

Potassium iodide .....	8.1
Fl. ext. lobelia .....	0.72
Fl. ext. hyoscyamus .....	0.72
Glycerin .....	12.0
Aromatic elixir .....	100.0
Sig.: One teaspoonful every three hours.	

If iodide intolerance occurs as noted by papular rash or pain in the salivary gland, ammonium chloride may be substituted.

3. *Aminophylline*.—Aminophylline acts directly on the bronchial musculature which it dilates. In doses of 0.24 to 0.48 gm. in 200 to 300 cc. of 10 per cent glucose it may be used intravenously once or more daily. A feeling of warmth sometimes develops during or immediately after its administration, and occasionally nausea and vomiting occur. The dose should be reduced and given more slowly and discontinued if vomiting persists.

4. *Atropine*.—Atropine has been advocated to inhibit vagus effect and thus release bronchospasm. However it has a drying effect which is seldom advisable, since liquefaction of bronchial secretion is the aim of therapy. *Stramonium*, a drug of the atropine group with similar action, is combined with potassium nitrate in ratio of 1 to 2. Inhalation of the smoke from this mixture is effective in relieving asthmatic paroxysms.

PROTECTION AND ELIMINATION.—To procure more permanent relief the patient must be protected from the causative agent or allergen. *Environmental control* alone is often sufficient to relieve the patient who may have been sick for many days. Many patients show dramatic improvement in twenty-four hours if they are taken to a hospital, where protection can easily be obtained from the inhalant, ingestant and contactant allergens and from physical factors. This is accom-

as tremor, palpitation and headache, and add to the apprehension of the patient. When an emergency demands epinephrine, there are few contraindications to its use. Some caution must be observed where there is complicating heart disease, especially coronary involvement. Some patients experience such extreme side-reactions of nervousness, palpitation and general weakness that care must be observed to use small doses and then only in emergency. Very occasionally, hypersensitivity to epinephrine has been reported similar to that occurring with insulin or other extracts of endocrine products. The crystalline form is well tolerated in these cases.

Epinephrine is not habit-forming. Patients may become refractory to it temporarily during severe and prolonged attacks of asthma, particularly in status asthmaticus. In such cases, after the use of a general anesthetic, epinephrine again attains its effectiveness. Sometimes when the hypodermic method does not give relief the intravenous administration is efficient. Dilution is increased to 1:10,000 for use by vein; or a slow venoclysis of saline or 10 per cent glucose containing 1 or 2 cc. of 1:1000 epinephrine in 500 cc. of solution may be given.

It is better for a patient to receive epinephrine in small amounts before an attack is well established, when larger quantities may be required to attain the same effect. The *adrenalin spray* is useful as a patient can administer the drug to himself as soon as he feels an attack starting. This is given by placing a solution of 1:1000 epinephrine in an all-glass atomizer, which is specially designed to give off the liquid in a fine spray. Holding the glass nozzle in his mouth, the patient squeezes the bulb of the atomizer and at the same time inhales. Epinephrine will decompose when exposed to light or air, or when in contact with metal. After epinephrine administration, skin reactions are inhibited for about one hour.

*Epinephrine in oil* is used to prolong the effectiveness of the drug. It is given intramuscularly and its effect lasts for six or eight hours. This slow-acting epinephrine is also used in 1:500 solution with a gelatine base.

2. *Potassium Iodide*.—Potassium iodide is the drug par excellence for liquefying bronchial secretion. This should be

apprehensive and requires sleep. Large doses of *barbiturates* may be used, as 0.6 gm. of barbital repeated once in two hours if necessary, or 0.2 gm. of phenobarbital in one dose. *Paraldehyde* is preferable since it is most effective in producing rest. It may be given by mouth or rectally. The dose rectally is 10 cc. in 60 cc. of olive oil or milk and may be repeated once or twice in twelve hours. When paraldehyde fails, general anesthesia must be tried. *Avertin* may also be used in doses of 60 mg. per kilogram of body weight. *Ether* may be used rectally in olive oil, about 75 cc. to 100 cc. of each. If asthma persists during sleep, *aminophylline* in glucose may be repeated after four to six hours. *Epinephrine* may be tried at intervals to test response. It should not be pushed too hard lest it act as a club rather than a benefit to these patients. *Iodides* must be pushed and if not taken by mouth may be given intravenously in 10 per cent solution.

*Oxygen*.—If oxygen is available it should be used just as soon as control by epinephrine and aminophylline is lost. Concentration of 40 per cent oxygen at the rate of 4 to 6 liters a minute as needed reduces cyanosis and renders breathing more comfortable. If *helium* can be used in ratio of 80 per cent helium to 20 per cent oxygen, improvement in dyspnea is quicker than when oxygen is used alone. Barach suggests the intermittent inhalation of 20 to 30 per cent oxygen and remainder helium, for forty-five-minute periods, three to five times daily. Better effect is obtained by preceding and following the gas with adrenalin spray.

In the great majority of cases, oxygen, aminophylline, paraldehyde and epinephrine will relieve status asthmaticus. In desperate situations *epinephrine* may be given slowly *intravenously*, 0.1 cc. of 1:10,000 solution, diluted with 1 cc. of physiological saline. It may be dangerous by affecting the heart, causing auricular fibrillation with resulting pulmonary edema. If the pulse is very rapid this procedure is contraindicated.

*Bronchoscopy* has been advocated as a therapeutic measure. At times it seems to be of value, especially if plugs of mucus can be removed.

*Morphine* is used extensively. It is not advised, though it

plished by stripping the room of all objects which cannot be kept dust-free, such as rugs, rug pads, drapes, overstuffed furniture and eiderdown comforts. Pillows and mattress should be covered with an impervious material; cotton blankets should be substituted for wool; and dust and pollen entering through the windows should be controlled with ventilators or filters. *Dietary control* is also necessary, since foods are an important group of allergens. The patient is placed on a basic diet consisting of simple foods that he does not have in his usual daily diet, excluding particularly the common offenders, milk, egg and wheat.

Patients who do not respond to environmental control as outlined above usually have intrinsic asthma.

#### PROLONGED ASTHMA OR STATUS ASTHMATICUS

The patient with asthma that lasts for a day or two or for many days may become an emergency in that he does not respond to the measures previously outlined. He becomes a distressing problem as he wheezes and pants for breath, unable to eat, rest or breathe. The prognosis is not so promising as in the acute uncomplicated asthma which responds to epinephrine. This drug often gives no relief in these chronic cases which are temporarily adrenalin-fast.

Any patient whose asthma is not properly treated may lapse from paroxysms of acute attacks into this state of continuous asthma. Chronic purulent bronchitis, bronchiectasis and emphysema occur as complications and produce this condition. Untreated sinus infections, often secondary to a neglected case of nasal allergy, predispose to chronic asthma. However, most cases have no detectable etiology and these are usually the severe expressions of intrinsic allergy. Many remedies have been advocated but none has been successful in all cases.

**TREATMENT.**—Besides adequate protection and the administration of epinephrine, potassium iodide and aminophylline, as previously outlined, patients with status asthmaticus should receive sedatives which give relaxation and restore effectiveness of epinephrine. The patient is usually worn out and

Before prescribing this, the patient should be asked if he has taken aspirin without mishap. *Neosynephrin* in 0.25 per cent solution and *propadrine* are also effective, on topical application, in reducing edema. Propadrine may also be taken internally, 24 mg. being given every three hours with effects equal to ephedrine and with few side-reactions. *Atropine* is sometimes necessary when the discharge is troublesome. The dose is 0.4 mg. once or twice a day. It causes dryness of the mouth. *Belladonna* is just as effective and is a constituent of U.S.P. *rhinitis tablets*. One of these, full strength, taken every fifteen minutes until nasal discharge is relieved, will control the discharge with less discomfort than atropine.

*Environmental Control.*—The ideal treatment is the avoidance of contact with the allergens. This entails, in the case of pollen allergy, remaining in an air-conditioned room or going to some locale where that particular pollen does not occur. For other types of inhalants, methods of avoidance will vary with each one. For food sensitivity, elimination diets are necessary.

*Specific Treatment.*—Hyposensitization may be accomplished where avoidance is not practical. Usually, injections for pollen hay fever are given before the season (preseasonal treatment), or perennial treatment may be carried through the year. However, for acute hay fever during the season, *coseasonal treatment* is successful in some cases though it does not give as good results as preseasonal treatment. Very small amounts of extract are given frequently. The patient is tested intradermally with 1:10,000 dilution or higher and the subcutaneous dose is chosen from the test giving a one or two plus reaction. The first injection will be 0.1 cc. subcutaneously of the selected dilution (for example, 1:10,000). Injections are repeated once daily, increasing each dose by 0.1 cc. until 0.5 cc. is reached (after four days), when 0.05 cc. of 1:1000 dilution is used and increased to 0.1 cc. where it is maintained. The interval is increased to every two or three days. If there is improvement the treatment is continued for three weeks. If, after six injections, there is no improvement, there is no use in continuing.

Another way of employing coseasonal treatment is by the

does allay apprehension and promote rest. In experimental animals it causes bronchoconstriction and, of course, it soon becomes habit-forming to many asthmatics.

### ALLERGIC RHINITIS (HAY FEVER)

When the shock organ is in the nasal mucosa, there result symptoms of violent sneezing, extreme obstruction to nose, copious watery discharge, itching of the nose and general symptoms of lassitude, weakness, malaise and alternating sensations of heat and cold. Some patients have considerable prostration. Sleep may be impossible; appetite impaired. Cough is often present especially at night, and there may actually be mild asthma. As in asthma the causes may be extrinsic or intrinsic.

#### Extrinsic Rhinitis

The extrinsic cases of seasonal occurrence are due to pollens. However, foods and inhalants such as orris root, feathers, animal epidermis, wool, house dust and other dusts met with in various occupations cause symptoms perennially.

**TREATMENT.**—Treatment is directed against the edema of the nasal mucosa which results from stimulation of the cholinergic nerves.

**Drugs.**—*Epinephrine* may be used as a topical application by spray or drops, but the side-effects do not justify the results. *Ephedrine* is of longer action and may be applied directly to reduce the edema and open the airway. Nose drops containing 1–2 per cent ephedrine in saline or oil may be instilled into the nose as often as every two hours if necessary. The patient can do this himself, preferably while lying on bed with head held back over side of bed. The physician can best do it by placing cotton swabs dipped in ephedrine solution in the patient's nostrils. Ephedrine is also effective by mouth, for which use a convenient prescription is:

Ephedrine sulfate .. . . . . .	0.015
Amytal . . . . .	0.03
Aspirin .... .	0.18

Sig.: One capsule every four hours.

sets, urticaria or angioneurotic edema especially of the lips and tongue, or other allergic manifestations. X-ray examination of the gastro-intestinal tract will reveal marked muscle spasm.

**TREATMENT.**—*Epinephrine* may be given and if very definite relief occurs an allergic cause is indicated. *Atropine* in 1 mg. doses hypodermically may also be tried. *Withdrawal of food* and the giving of only *parenteral fluids* will help to allay symptoms. A *purge* will remove the allergen more quickly from the gastro-intestinal tract but, of course, should not be given if there is obstruction or suspicion of appendicitis.

### URTICARIA (HIVES) AND ANGIONEUROTIC EDEMA

Urticaria and angioneurotic edema are due to the same process of edema varying only in degree. The acute type results from foods, drugs, contactants and occasionally inhalants. Repeated exposure to these offending substances may cause a semblance to chronicity. However, the great number of cases of urticaria are probably not due to atopy, but are intrinsic in character, wherein such factors as foci of infection, endocrine or emotional disturbances and physical agents such as heat, cold, sunlight and friction release a display of atopic-like disorders. Emergencies may arise due to the location of the swelling, particularly if in the throat and larynx with danger of suffocation. In such cases intubation or tracheotomy may be necessary.

**TREATMENT.**—As in other types of allergy, *epinephrine* is the most efficient drug. If necessary it may be injected into a local lesion to reduce the edema. Topical application as with a spray may supplement injection. Urticaria causes intense itching and discomfort when generalized and seems to become refractory to epinephrine, with the result that the patient becomes exhausted from lack of rest, nervousness and poor nutrition. These patients resemble those with exhaustion from intractable asthma. They require heavy *sedation* and procedures similar to those used in status asthmatica to give rest. Improvement often follows such measures. *Colloidal baths*, application of *calamine and zinc lotion* containing 1 per cent phenol and *cold applications* give considerable relief from

*intradermal method.* Each day the patient receives an intradermal injection of an amount of extract that will give local skin reaction, using the dilution of extract to which the patient first reacts. The amount in one site should not exceed 0.1 cc.; and, if more is given at a dose, several injections should be made. Thus increments would be 0.1, 0.2, 0.3, 0.4 and 0.5 cc. of, for example, 1:10,000 dilution (or higher dilution if the patient is very sensitive), and then 0.05, 0.1 and 0.2 cc. of 1:1000 dilution. The dose is judged by some allergists by the intradermal reaction which is about the size of the thumb-nail. This quantity is maintained regardless of the strength of dilution. With the intradermal method the margin of safety is narrow. Local reactions are more uncomfortable and systemic reactions more frequent. Relapse with coseasonal treatment may be due to overdosage or too long intervals between injections.

### Intrinsic Atopic Rhinitis

As in intrinsic asthma the cause is not demonstrable by control of environment. Intrinsic factors cause numerous cases of allergic rhinitis of extreme severity. These patients are usually in middle life. A low basal metabolism is often present.

**TREATMENT.**—Acute symptoms are treated by drugs used for the extrinsic type to combat edema and increased vagus-tone. *Theelin* and *thyroid* are sometimes effective. *Histaminase* in large doses of 75 to 100 units daily may be worthwhile. Injections of *histamine* and *acetylcholine* are also used. *Ionization* may be helpful in some cases. *Cauterization* of the nasal mucous membrane and especially *submucous diathermy* may give considerable relief.

### GASTRO-INTESTINAL ALLERGY

Owing to marked edema of the mucosa of the gastro-intestinal tract and the muscle spasm, allergic reactions may simulate acute surgical emergencies, especially intestinal obstruction and acute appendicitis. The allergen is usually a food. *Diagnosis* may be very difficult and the decision whether to operate or not may have to be made. History must be carefully taken for occurrence of repeated gastro-intestinal up-

*Technic for Intravenous Injections of Histamine.*—The intravenous method entails the slow injection of 0.2 mg. or 0.2 cc. of 1:1000 dilution of histamine diphosphate diluted to 20 cc. with physiological saline solution. A small needle is used and the histamine solution is injected a little at a time, with short pauses between injections. The patient complains of warmth, flushing of the face and headache. The injections are discontinued until these symptoms disappear, then resumed more slowly. Speed of administration is determined by the patient's reaction which should be minimal. The next day the dose is 0.4 mg. and on the fourth day 0.6 mg. Usually there is a decrease of the itching and in the number of hives after the first or second injection.

*Schedule for Subsequent Subcutaneous Injection.*—The intravenous injections may be followed by subcutaneous administration according to the schedule on page 438.

#### PHYSICAL ALLERGY

This term, proposed by Duke, implies production of allergic disorders under the stimulus of physical agents such as heat, cold, light and mechanical irritation. The chief manifestation of physical allergy is urticaria. Through stimulation by physical agents, histamine or H-substance is released, and whealing or massive edema results. When large amounts of histamine are released suddenly into the blood, severe reactions occur. Instances of sudden collapse while swimming suggest that hypersensitiveness to cold with resulting shock might be the cause of drowning in many cases.

*TREATMENT.*—An attempt may be made to *desensitize* persons who have cold hypersensitivity if such is known. This may be done by subjecting the patient to decreasing temperature baths starting at 90° F. A daily bath is taken at temperatures two degrees lower than on the previous day, until the patient can stand 65° F. without symptoms. A daily cold shower is taken to keep up the desensitization. Similarly, a heat-sensitive person may start with a cool bath and each day increase the temperature until he tolerates temperatures of 100° F. or higher. It is important in such therapy that the patient be immersed in the bath suddenly. Rubbing the skin

the itching. Protective measures are also instituted to eliminate extrinsic agents and give protection against food, inhalant and contactant.

Treatment of the chronic, intrinsic type of urticaria, aside from the symptomatic treatment, is aimed at possible internal causes. *Theelin* is given with good results in cases of urticaria occurring at the time of the menopause; *psychotherapy* seems to have a definite beneficial effect; *histaminase* has been used in large amounts in these cases with varying effect. However treatment with *histamine* injections gives more favorable results. The rationale of this procedure is vague. The drug is administered by iontophoresis into the skin, by subcutaneous injection, or intravenously.

SCHEDULE FOR SUBCUTANEOUS INJECTIONS OF HISTAMINE

Injection	Dilution	Amount, Cc.	Interval
1	1:10,000	0.05	Twice a day
2	"	0.05	
3	"	0.10	
4	"	0.10	
5	"	0.15	Once a day
6	"	0.20	
7	"	0.25	
8	"	0.30	
9	"	0.35	
10	"	0.40	
11	"	0.45	
12	"	0.50	
13	1:1,000	0.05	Every 2 days
14	1:1,000	0.10	
15	"	0.15	
16	"	0.20	
17	"	0.25	
18	"	0.30	
19	"	0.30	
20	"	0.30	Every 3 days
21	"	0.30	
22	"	0.30	
23	"	0.30	
24	"	0.30	

den distress, apprehension, seems unable to talk, may gasp or cry out, turn ashen gray and sink into coma. Death may come rapidly. However, various allergic manifestations such as suffusion of the face, generalized itching, urticaria, asthma, and gastro-intestinal symptoms like vomiting and diarrhea may occur. If these latter signs appear before shock, the patient is less likely to suffer a quick disaster.

**TREATMENT.**—In order to delay further absorption of antigen, a *tourniquet* is applied to the arm in which the injection was given. *Epinephrine* 1:1000 in doses of 0.5 to 1 cc. is given at once subcutaneously into another extremity. If the injection of antigen has been given intradermally, epinephrine is injected into the site and just above it to delay lymphatic drainage from area. *Cold compresses* may be applied to local reactions. Intravenous administration of epinephrine 1:10,000 may be indicated in shock. The dose should be small, 0.2 to 0.3 cc. Patient is kept lying quietly wrapped warmly. *Artificial respiration* may be necessary and *oxygen* given if it is available.

#### Precautions

The injection of an antigen for *desensitization* should be given deep subcutaneously into an extremity. Before the plunger is pressed down it should be withdrawn to make certain that the needle is not in a vein. Again it must be emphasized that the dose and interval must be properly adjusted and no short cuts taken. Epinephrine mixed with an extract delays absorption, but a reaction no less severe may take place later when the patient is out of reach of the doctor. Exercise or heavy exertion within several hours after an injection may help to bring on a reaction. The patient should be instructed to carry capsules of ephedrine, each containing 0.024 gm., and at the onset of the first symptoms take a capsule and return at once to his physician. Usually the ephedrine is sufficient.

In *skin testing* it is safer in children to use the scratch method, especially for egg and pollen. Egg in minute quantity may produce violent reaction of systemic type or a shock-like collapse in a very sensitive person. Intradermal testing should not be done with egg antigen in a dilution of less than

briskly with a coarse towel may help release of histamine and thus reduce the skin reactivity to friction as occurs in cases of dermatographia, an accompaniment of urticaria.

#### PURPURA DUE TO ALLERGY

Allergic purpura, Henoch's variety, also known as the "erythema group with visceral symptoms," may present an acute crisis due to hemorrhage and also a complicating nephritis. The most effective therapy in such cases seems to be allergic control. Accompanying the purpura are usually urticaria, abdominal pain, arthritis, urinary findings such as hematuria, and gastro-intestinal bleeding. In the majority of cases foods are at fault. Skin testing is not of value but the use of *elimination diets* is the most effective diagnostic and therapeutic measure. However, inhalants have been known occasionally to cause trouble.

#### REACTIONS DUE TO PARENTERAL INJECTION OF SERA AND OTHER ANTIGENS

##### Local Reactions

When any parenteral injection of an antigen is given, danger of reaction is always present. At the site of the injection redness and edema may occur. The appearance of a local reaction may be immediate or delayed from several to twenty-four hours. It is most commonly met with during the performance of skin tests. A wheal appears, surrounded by a flare of erythema. Gradually edema becomes more pronounced. Urticaria may appear.

##### Constitutional Shock

A generalized reaction of severe nature may take place within a few minutes after injection of an antigen or serum, usually with some signs of a local disturbance at the site of injection. Testing and treatment with undiluted horse serum and pollen extracts have caused most of the violent and serious reactions.

A patient who has a constitutional reaction experiences sud-

case desensitization is practically impossible; however, some other type of serum may be used, such as rabbit serum, employing the same preliminary tests. Undiluted rabbit serum gives a nonspecific reaction which is not present when the serum is diluted 1 to 10.

A patient with negative skin tests, who gives a history of receiving previous serum, or who has a history of asthma, may be treated by giving serum according to the desensitization schedule. A patient with positive skin tests but without history of atopy may be given serum very cautiously and desensitization may be possible.

The injections of serum are given deep subcutaneously at twenty-minute intervals according to the following desensitization may be possible.

Injection	Dilution of Serum	Dose, Cc.	Site
1	1:10	0.1	Arm
2	"	0.2	"
3	"	0.4	"
4	"	0.6	"
5	"	0.8	"
6	Undiluted serum	0.1	Other arm
7	" "	0.2	" "
8	" "	0.4	" "
9	" "	0.6	" "
10	" "	0.8	" "
11	" "	1.0	Thigh

If a reaction to any given dose occurs, treatment should be given similar to that described under "Constitutional Shock."

#### Intravenous Desensitization

In the administration of antipneumococcus and other therapeutic sera, the intravenous route is sometimes indicated. Administration should be begun by giving the serum at first subcutaneously according to the above schedule and then continued by giving the injections intravenously with a 26- or 27-gauge needle at twenty-minute intervals as follows:

1:1000, and preferably should be preceded by a scratch test. Pollens and other inhalants are safer for intradermal testing if used in 1:1000 dilution or weaker.

### Avoidance of Serum Reactions

Serious consequences from the administration of serum can be prevented by recognizing factors which may lead to trouble, by making preliminary tests, and by using proper methods of desensitization.

**HISTORY.**—Careful inquiry should be made into history of family allergy, personal allergic symptoms, previous serum inoculations and the kind of serum used, lapse of time since previous serum, and occurrence of serum sickness.

**PRELIMINARY TESTING.**—Preliminary testing should be done whether a history is negative or not. (1) *Scratch test* should be made with 1:10 dilution of the serum to be used. *Normal* horse or other serum should be used because tests with immune sera often produce the so-called E-E reaction which is nonspecific but may be confused with a positive allergic test. The scratch test with 1:10 dilution is the equivalent to an intradermal test with 1:1000 dilution. Tests are read in fifteen minutes. (2) *Conjunctival test* is done by using the same 1:10 dilution in physiological saline. A drop is placed in the inner canthus of the eye. A positive reaction is denoted by a suffusion of the conjunctiva. Test is read in five minutes. Conjunctiva should be rinsed with a mixture of 4 cc. of adrenalin 1:1000 in 12 cc. of boric acid solution. (3) *Intradermal test* is performed using the same 1:10 dilution and is read in twenty minutes. (4) As a final preliminary test an intradermal therapeutic dose of undiluted serum, 0.02 cc., is given and is watched for ten or fifteen minutes while preparing for treatment.

**ADMINISTRATION OF SERUM.**—In the absence of positive findings in any of these tests, serum may be given without delay. Nevertheless, caution should be exercised, using a safe method like that outlined below for desensitization. If a patient has a positive history of asthma due to horse dander and the cutaneous and ophthalmic tests are positive, the injection of therapeutic horse serum is contraindicated. In such a

## THE MANAGEMENT OF THE EMERGENCIES IN INFANCY AND CHILDHOOD

JOHN ZAHORSKY, M.D., F.A.C.P.\*

If we define an emergency as a combination of circumstances requiring immediate action or remedy, the question arises—who decides that such a condition exists? In pediatric practice obviously it is the child's attendant—parent or nurse. In most instances it is a symptom or a symptom complex that alarms the parents. Therefore, it is most satisfactory to group these emergencies under the headings of syndromes, which apparently require immediate relief and the diagnosis of which demands prompt investigation.

### CONVULSIONS

A convulsive seizure in a child is always a terrifying spectacle and the parents almost invariably become panic-stricken. Yet the immediate prognosis of a paroxysm occurring at the onset of an acute illness is, with few exceptions, favorable. The recognition of the paroxysm presents no difficulties and is usually clear at a glance. The staring eyes, the unconsciousness, the rigid body, the irregular respiration, the clonic spastic movements, the contortions of the face, the retraction of the neck and, finally, the violent jerkings of the extremities comprise a syndrome that can be mistaken for nothing else.

**TREATMENT.**—The rectal temperature should be taken at once. If the fever is very high ( $104^{\circ}$  to  $107^{\circ}$  F.) the child should be stripped and placed in a *tepid bath* ( $90^{\circ}$  F.). Either a hot or cold bath will increase the nervous excitability and the former is dangerous. A cold bath induces contraction of the peripheral arterioles and increases the congestion of the internal organs. The tepid bath may be entirely sufficient to quiet the nervous system. When little or no fever is present the bath may be omitted.

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\* Professor of Pediatrics and Director of the Department, St. Louis University School of Medicine; Pediatrician-in-Chief, St. Mary's Group of Hospitals.

Injection	Dilution of Serum	Dose, Cc.
1	1:100	0.1
2	"	0.2
3	"	0.4
4	"	0.8
5	1:10	0.1
6	"	0.2
7	"	0.4
8	"	0.8
9	Concentrated serum diluted with equal amount of saline	0.1
10	"	0.2
11	"	0.4
12	"	0.8
Rest of serum given slowly, diluted with equal amount of saline as a venoclysis		

### Serum Sickness

This is a delayed reaction that occurs following a first injection of serum after a period of incubation, usually of seven to ten days. This occurs in the large percentage of persons receiving serum. It is an uncomfortable inconvenience to the patient but is not fatal and is self-limited. The treatment is symptomatic. Histaminase may be given in large doses at about the time that symptoms are expected to appear.

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If the jerkings persist, an *anesthetic* should be administered. For this purpose, nothing is so efficient as the inhalation of enough chloroform to quiet the jerkings and produce a relaxation of the rigid trunk muscles. Ether is not a good substitute, because it temporarily increases the nervous excitability. As soon as a reasonable respiration is established, the anesthetic should be discontinued. If the convulsion reappears in a few minutes, the chloroform treatment may be repeated. In very stubborn cases I sometimes resort to a hypodermic injection of *morphine* ( $\frac{1}{40}$  grain for an infant one year old). However, a medicated *retention enema*, after a cleansing enema, is often effective (3 to 5 grains of chloral hydrate in 1 ounce of water or 2 grains of barbital in cocoa butter). Both chloroform and chloral hydrate have a tendency to produce an acidosis which is really advantageous to these infants, as an alkalosis increases the nervous tension.

In recent years medication by the *barbital preparations* has been extensively tried in the treatment of convulsions, although following my own experience I still prefer chloroform and chloral hydrate. Nevertheless, the hypodermic administration of sodium amytal ( $\frac{1}{2}$  grain for an infant one year old, 1 grain for a child five years old) is a potent remedy for quieting the child. The hypodermic injection of magnesium sulfate is not often indicated, since its paralyzing effect may decrease the child's resistance to disease.

DIAGNOSIS.—As soon as the child is asleep after an eclamptic seizure, the diagnosis of the pathologic process behind the syndrome becomes an urgent task. As the majority of cases are accompanied by *fever*, this symptom becomes the diagnostic problem. A careful physical examination fortified by a study of the blood and urine is necessary. An infectious sore throat is undoubtedly the most frequent cause of convulsions in infancy, and is readily recognized. Pneumonia, dysentery and any acute febrile disease (scarlet fever, malaria, etc.) may be ushered in by a convulsion. However, the urgent problem is to exclude intracranial diseases. If the fever persists, the rigidity of the neck does not completely relax after the child has slept, and Brudzinski's sign appears, a *lumbar puncture* is indicated. This serves not only as a diagnostic

procedure but also is a powerful therapeutic measure to diminish the intracranial pressure. In all stubborn cases of convulsions, meningitis and encephalitis must be excluded by the physical examination aided by an examination of the spinal fluid.

Whenever the history suggests a possible acute intestinal disease, the *stool*—usually liquid in these cases—should be examined for pus cells. An acute enteritis may thereby be revealed many hours before a manifest diarrhea develops.

When little or no fever is present the diagnostic survey should include chronic disease of the brain—epilepsy, tumor, hydrocephalus—also metabolic disturbances—alkalosis and diabetes. A blood examination is needed to exclude hypocalcemia. Finally, it should not be forgotten that convulsions may be produced by uremia—rather rare in infancy.

The treatment thereafter is adjusted to the diagnostic indications: the sulfonamide preparations for pneumonia or meningitis, calcium gluconate for alkalosis and dextrose for hypoglycemia.

#### GROUP

Although the term “croup” has been deleted from the list of respiratory diseases, it is useful to the practitioner as a name for a common and sometimes dangerous syndrome. The *chief symptoms* are hoarseness, a barking or rasping cough, laryngeal stridor and a varying degree of dyspnea. The symptoms may come on gradually, but the most alarming forms of this disorder arise suddenly at night. The child awakens abruptly with a hoarse, strangling cough and the breathing is accompanied by a rasping tone, which has a distinct quality easily recognized by the experienced physician.

**TREATMENT.**—In all probability some viscid mucus, more or less desiccated, is clinging to the laryngeal mucous membrane, and in addition there is a spasm of the intrinsic laryngeal muscles. The quickest way to relieve the symptoms is to *induce vomiting*. A teaspoonful of syrup of ipecac or the same dose of compound syrup of squill by mouth will cause vomiting within fifteen to twenty minutes, after which the child will go to sleep and breathe easily, although a slight stridor may persist for several hours. However, if the symptoms are

comparatively mild the emetic should be omitted; one may try *grease* or *oil*. A teaspoonful of butter, lard, olive oil or even mineral oil may be administered every few minutes. Two other measures should be instituted immediately. One is the application of a *cold compress* to the larynx and trachea, the other is the utilization of *hot vapor inhalation*. For the latter purpose the teakettle may be used, but a more efficient device may be bought at the drugstore (croup kettle, benzoin vaporizer, electric vaporizer). It is really not necessary to add a medicament to the steaming water, although there is no objection to the use of compound tincture of benzoin or menthol. Having relieved the alarming symptoms the next task is to make a diagnosis.

**DIAGNOSIS.**—Acute catarrhal laryngitis, or spasmodic croup, is a symptom of many different diseases, and successful treatment depends largely on an exact diagnosis. It is seen in the *common cold* and evidence of the presence of a cold should be sought in the history of the patient, and in the family. A painstaking inspection of the nose and throat is necessary.

The catarrhal changes may seem obvious, but one must always be on the lookout for a *diphtheritic exudate*—in the nose, on the tonsil, and especially on the lateral walls of the pharynx. The soft palate should be raised with the tongue depressor to give access to the nasopharynx. Even a small exudate no larger than a pea arouses the suspicion of diphtheria. If the dyspnea and laryngeal stridor persist for several hours, it is a good practice to inject a therapeutic dose (10,000 units) of antidiphtheritic serum as a precautionary measure. Of course, a throat culture is vitally important in all cases of croup which do not yield to the simple treatment.

A large dose of antidiphtheritic serum (15,000 to 20,000 units) is necessary if clinical or bacteriologic evidence of diphtheria is discovered. The necessity of intubation depends on the length of the illness and the severity of the laryngeal obstruction. Aspiration of the larynx is now successfully practiced, and only in badly neglected cases does tracheotomy or even intubation become an emergency procedure.

Another serious disease which is accompanied by severe and stubborn symptoms of croup is *subglottic laryngitis*—a

disease known for a century but which has received much study in recent years, and is now called "acute laryngotracheobronchitis." The bacteriologic data have not been clearly elucidated. We know that a severe inflammation with great swelling of the subglottic mucous membrane and even a fibrinous deposit occurs in these cases. A definite diagnosis can be made only by laryngoscopy. The practitioner is compelled to make such a provisional diagnosis in stubborn cases whenever diphtheria has been excluded by bacteriologic studies and by therapeutic tests.

Contrary to some recent statistics we have as a rule found the prognosis in subglottic laryngitis to be favorable. The old treatment with iodized calcium has been replaced by use of the sulfonamide preparations. We employ a continuous humidification of the room for several days. One-half tablet (each 0.5 gm.) of sulfathiazole or sulfadiazine is administered by mouth every four hours to a child weighing 20 to 30 pounds. Intubation and tracheotomy are dangerous and these operations should not be performed except as a final resort in desperate and neglected cases.

In the differential diagnosis of acute croup one must also consider three other conditions, namely a *foreign body* in the larynx, *edema of the larynx* and *hypocalcemic laryngospasm* (tetany).

#### SYNCOPE

Attacks of fainting are rather uncommon in infancy and early childhood but more frequent in later childhood. The act of crying tends to inhibit the tendency to syncope, but any procedure which excites pain or provokes nausea may induce it. Curiously, diseases of the heart do not produce syncope, except the disorder characterized by an extreme bradycardia (heart block). As a prophylactic procedure it is advisable to place all children on whom some simple operation is to be done in the recumbent posture.

TREATMENT.—The treatment consists in placing the child in a recumbent position with the head low and giving a dose of a diffusible stimulant—coffee, whiskey, spirit of camphor or aromatic spirit of ammonia. The hypodermic injection of a

solution of adrenalin chloride—4 to 5 minims—is rarely necessary.

**DIAGNOSIS.**—The symptoms of syncope are well known; nausea, dizziness and pallor are the premonitory manifestations. Then the child falls and becomes semiconscious. The pulse is feeble and usually very slow. The respirations are also shallow and slow. The blood pressure falls, sometimes to an alarming degree. The muscular system remains relaxed and no muscular jerkings are observed.

The *differential diagnosis* must consider eclampsia, heart failure and shock. When the physician observes the patient during a fainting attack the diagnosis presents no difficulties. However, in obtaining the history of one or more previous attacks the description of the symptoms by the parent are usually too indefinite to separate syncope from minor epilepsy.

#### CARDIAC FAILURE

Sudden heart failure in children is comparatively rare, except in diphtheritic myocarditis. Even in this disease, premonitory symptoms point to a failing circulation a few hours before death. Acute cardiac dilatation from rheumatic myocarditis is sometimes the cause of an emergency. Rarely an intestinal intoxication produces a serious form of myocardial disease, the symptoms of which are tachycardia with falling blood pressure. The occurrence of coronary thrombosis is almost unknown during childhood. We have seen sudden death occur in extreme mitral stenosis and in chronic adhesive pericarditis. Attacks of paroxysmal tachycardia are manifestly alarming but rarely end fatally. Congenital heart disease increases the liability to paroxysmal attacks of cyanosis and dyspnea.

**TREATMENT.**—Digitalis is contraindicated in the bradycardia and the late circulatory failure following diphtheria. Restlessness should be relieved by the hypodermic injection of *morphine* ( $\frac{1}{16}$  grain to a child of five years). As a profound metabolic disturbance is associated with the circulatory failure and vomiting is almost invariably present, the intravenous injection of *glucose* is indicated (10 per cent solution). The infusion should be given very slowly over a pro-

longed period of time. Small doses of *epinephrine* may be administered hypodermically every half hour. Camphor and strychnine, formerly used by practitioners, have been generally abandoned. Sometimes a slight improvement of the pulse may be obtained by the use of *caffeine* (*caffeine sodio-benzoate* solution, dose 1 to 2 grains).

For the congestive heart failure of rheumatic fever, *digitalis* is indicated. Acute cardiac dilatation from *mitral stenosis* or adhesive pericarditis sometimes is temporarily improved by a hypodermic injection of a *digitalis* solution (I have used *digitolin*, 0.5 cc., for a child 5 years old). Acute decompensation of the heart induced by any severe infectious disease is rarely benefited by the administration of *digitalis*. This drug may be tried in the syncopal attacks of congenital heart disease, if tachycardia, not bradycardia, is manifest.

It is difficult to relieve the symptoms of *paroxysmal tachycardia* promptly. I have prescribed *quinidine sulfate* (1 grain every three hours) with apparent benefit, but have had no success by pressure over the carotid arteries. Recently, *mecholyt*—dose 2 to 5 mg. hypodermically, has been recommended in these cases.

I have had no experience with *venesection* to relieve a dilated heart. On physiological grounds it seems indicated in selected cases. The administration of *oxygen* is an essential part of the restorative treatment.

#### RESPIRATORY FAILURE

An emergency frequently arises when a child does not receive a sufficient amount of oxygen into the blood (*anoxemia*) to meet respiratory requirements. The obvious symptom is difficulty in breathing, or failure to breathe. The physician at a glance should decide whether the respiratory movements are feeble or labored. If definite cyanosis is present the immediate inhalation of *oxygen* is indicated and the central nervous system may be stimulated by the hypodermic injection of *caffeine sodio-benzoate* (dose 1 grain) or *alpha-lobeline* (dose 5 to 10 minims of the solution).

DIAGNOSIS.—We distinguish two forms of respiratory failure, the obstructive and the paralytic. The former is due to

an obstruction in the respiratory tract, the latter is caused by a central or peripheral lesion in the nervous system. It is beyond the scope of this article to discuss accidents, such as drowning, the aspiration of foreign bodies, accidental hanging, or the rupture of a varix or abscess into the trachea. The management of croup has been discussed.

*Bronchiolitis of Young Infants.*—Feeble infants sometimes fail to expel the viscid mucus that accumulates in the bronchioles during an attack of bronchitis. This may be complicated by a lung involvement (bronchopneumonia). As a rule the infant shows little or no fever, but the labored respiration accompanied by a persistent cough points to a pulmonary disease. Auscultation reveals subcrepitant rales over both lungs and a very feeble respiratory murmur.

We sometimes resort to an *emetic* in these cases. The vomiting expels some of the mucus from the bronchial tubes. For such infants, a *mustard pack* is applied to the chest, and *steam inhalations* are used for several hours. The inhalation of *oxygen* will dispel cyanosis. The strength of the infant should be maintained by regular feeding. *Whiskey* is useful to quiet the cough (10 to 20 drops every two hours). *Expectorants* in small doses may hasten the liquefaction of the mucus. *Syrup of ipecac* in 10-drop doses may be given every three hours.

*Asthma.*—Asthmatic attacks may occur in infancy but the incidence is much higher during childhood. The sudden onset, expiratory dyspnea, sibilant rales and afebrile course are characteristic. On holding the ear close to the chest the asthmatic wheeze is generally perceived.

A hypodermic injection of *epinephrine hydrochloride* (5 to 8 minims of a 1:1000 solution) brings prompt relief in older children, but is less effective in infants. No untoward results from epinephrine have been reported, and one should not hesitate to give a full therapeutic dose. The dose may be repeated in an hour or longer. If sleep is necessary, 1 grain of *barbital* may be prescribed to a child of five years. However, for quick action, a subcutaneous injection of *codeine* ( $1\frac{1}{2}$  to  $\frac{1}{8}$  grain) is gratifying. It should be remembered that an asthmatic attack has a marked tendency to subside in two to three days, but recurrences are to be expected.

A rational diagnosis demands a search for the offending substance to which the child is allergic—often a difficult task.

*Bulbar Poliomyelitis.*—The most distressing complication and the most frequent cause of death in infantile paralysis is bulbar paralysis with resulting diaphragmatic immobility. The diaphragm does not contract and the child dies of suffocation. For many hours the child, by voluntary contraction of the accessory respiratory muscles, may manage to carry air into the lungs. Then sleep supervenes and respiration ceases. Artificial respiration carried on for weeks or months by means of the Drinker apparatus may save some of these unfortunate little patients.

More rarely a central paralysis of the diaphragm may be caused by *diphtheria* or *encephalitis*. A cessation of respiration due to a compression of the medulla following an *injury to the brain* is a dreaded accident that may baffle the brain surgeon.

Dangerous respiratory deficiencies, of course, may arise from a *massive pneumonia* or a *large pleural effusion*. In infants the respiratory difficulty may come on suddenly and unexpectedly, and the oxygen tent is urgently needed. In rapid pleural effusions, aspiration of serum or pus may be necessary. It should be remembered that sudden dyspnea and cyanosis may arise from a *pneumothorax*. Often a roentgenogram is necessary for making positive diagnosis. Aspiration of the pleural air is of doubtful value but may be employed to relieve extreme dyspnea; however, removal of too much air may again open the pulmonary lesion, causing the pleural cavity to refill with air. Dependence must be placed chiefly on oxygen inhalation. The lung lesion gradually heals and the air is absorbed.

#### EPISTAXIS

Most cases of nosebleed are relatively harmless, but severe and protracted cases arise which are the occasion for urgent professional calls.

*TREATMENT.*—If the nasal hemorrhage has lasted more than ten minutes in spite of simple treatment—rest, compression of the nose and cold applications to the face—a *tampon* (gauze

or cotton) moistened with a few drops of epinephrine solution should be inserted deep into the nose. A solution of ephedrine (1 per cent) may be used instead. Hydrogen peroxide, full strength, is a good hemostatic. A tamponade of the posterior nares is rarely necessary. If the bleeding continues, a hypodermic injection of *fibrogen* (1 cc.) may be tried. The intramuscular injection of human *blood*, 30 to 60 cc., is indicated in stubborn cases. Recently, *vitamin K* (Klotogen) has been successfully used in some stubborn hemorrhages, although its curative value is greatest in true hypoprothrombinemia, as in the hemorrhagic disease of the newborn.

**DIAGNOSIS.**—The cause of a spontaneous nasal hemorrhage usually may be determined from a study of the history. As a rule, the temperature should be taken, as severe nasal bleeding may be the initial symptom of an infectious disease, as measles, influenza, or typhoid fever. Inspection of the nares and throat to discover a foreign body or a possible diphtheritic exudate is part of the routine examination. A passive congestion from a disease of the heart sometimes produces a serious blood loss from the nose. Uremia, by increasing the blood pressure, may be the cause of recurrent epistaxis. It should not be forgotten that severe epistaxis may occur early in thrombocytopenic purpura and in leukemia. In a young child the possibility of scurvy should be considered. In many cases a small ulcer of the septum may be discovered which is treated by cauterization (nitrate of silver, chromic acid or the actual cautery). Fortunately, most cases of epistaxis are due to traumatism or any excitement that increases the blood pressure and not much blood is lost.

#### INTESTINAL INTOXICATION WITH DEHYDRATION

The symptom-complex known as dehydration or anhydremia which occurs in intestinal intoxication is not so frequently met today as twenty years ago. Most cases of intestinal intoxication arise from food poisoning or gastro-enteritis, diseases accompanied by vomiting and diarrhea. The baby becomes semiconscious and the skin assumes an ashy hue. The eyes appear sunken and the fontanel depressed. The skin is dry and, when pinched, reveals a loss in elasticity. The respira-

tion varies, depending on the degree of acidosis. The urine is almost completely suppressed, while the tissues are continuously depleted of water and salts by the watery diarrhea. As a rule, the fever is not very high and may be subnormal.

**TREATMENT.**—When the baby is seen early in the home, an attempt should be made to supply the necessary fluid and salts by simple means. I still adhere to the old treatment. We prepare *barley water* (or rice water) with 3 per cent barley flour and 5 per cent sugar (cane sugar or dextrimaltose) and 0.3 per cent common salt. According to the age of the infant, 3 to 6 ounces of this barley water should be fed every three hours day and night. It is really surprising how rapidly the serious symptoms subside after this simple treatment. I have not been impressed by the results of substitution of apple powder for the barley and sugar.

When the infant has been neglected and the intoxication has persisted for a day or more, it is expedient to hospitalize the infant. The lost fluid and salts are replaced by *hypodermoclysis*. For this purpose I usually prescribe equal parts of Ringer's solution and a 6 per cent solution of glucose. Several ounces may be supplied to the body in this way. A physiological salt solution may be substituted for the Ringer's solution. If symptoms of acidosis are manifest, Hartmann's solution is to be preferred. The *intraperitoneal injection* of salt solution is not often prescribed, as an element of danger is added by this procedure. Continuous *intravenous therapy* may be necessary in serious cases. We favor the technic of Ashly and Moore (J. Ped., 6: 88, 1935).

In passing, we may refer to the old treatment formerly used in cholera infantum: a hypodermic injection of morphine and atropine. This is a powerful remedy for checking the tremendous loss of water from the intestine and may be tried in selected cases.

**DIAGNOSIS.**—It must be admitted that diagnosis in gastrointestinal diseases has not been satisfactorily elucidated. We try to place such cases in the categories of food poisoning, diarrhea of the newborn, infectious enteritis, bacillary dysentery, parenteral infection and allergy. The presence of microscopic pus in a watery stool is a sign of an infectious enteritis.

Of course, a bacteriologic diagnosis should be attempted in all forms of enteritis.

### ACIDOSIS

As a rule acidosis—that is, a shifting of the ion concentration of the blood to the acid side—is accompanied by dehydration, the result of diarrhea and vomiting. We have seen cases in which the signs of dehydration were insignificant and yet the child manifested serious symptoms: deep breathing, flushed skin, anxious countenance and acetonuria. This syndrome is sometimes encountered as a complication of acetonemic (cyclic) vomiting. More frequently a gastro-enteritis or dysentery causes the acidosis. Diabetes, of course, is a common cause of air hunger.

**TREATMENT.**—Often a gastric sedative (phenol,  $\frac{1}{20}$  minim in milk of magnesia,  $\frac{1}{2}$  dram) and the oral administration of water containing sodium citrate (0.5 per cent) and sugar (7 per cent) will allay the symptoms. In severe cases we use Hartmann's solution by prolonged hypodermoclysis. A venoclysis of Hartmann's solution with glucose is indicated in the dangerous forms of this metabolic disturbance. Much depends on the promptness in recognizing the disease. In the cases due to starvation a few doses of sodium citrate (10 grains every hour) will rapidly allay the hyperpnea and mental distress if gastric feeding can be instituted.

**DIAGNOSIS.**—It is not always easy to make a comprehensive diagnosis in these cases. Deep and rapid breathing may be caused by diseases of the heart or lungs. Furthermore, a paralysis of the diaphragm or intercostal muscles (poliomyelitis, diphtheria) may cause unnatural respiratory efforts. In all cases the question of diabetes mellitus presents itself. A specimen of urine should be obtained—by catheterizing the bladder if necessary—and tests made for sugar and diacetic acid. When sugar is present in the urine it is best to make a determination of the blood sugar also. The ketosis of diabetes is treated with insulin, glucose and water.

Clinically, measurements of the hydrogen ion concentration of plasma are rarely made. We must depend on the clinical signs and symptoms and the exclusion of organic diseases of the heart, lungs and kidneys. In periodic attacks of cyclic

vomiting, alkalosis might be expected, but an acetonemia and ketonuria regularly occur. Sedatives, sodium chloride and sugar are the most effective agents for treatment.

Some confusion has arisen in the differential diagnosis of acidosis and alkalosis. It is assumed that protracted vomiting induces an alkalosis rather than an acidosis, owing to loss of hydrochloric acid. However, in infants there is so little secretion of the acid into the stomach that this assumption must not be taken seriously. The systemic depletion of carbohydrates from starvation will generally overbalance the trifling loss of hydrochloric acid. Clinically, we depend on the type of breathing, acetone breath and acetonuria for diagnosis.

### COLIC

Many babies cry excessively during the first three or four months of life. I favor the theory that these infants belong to a distinct constitutional type—the neurotic type, characterized by hyperexcitability and hypertonicity. Recently it has been shown by roentgen studies that the intestine of the young infant presents a deficiency pattern, similar to that found in avitaminosis. This indicates a disturbed or inefficient nerve function characterized by hypermotility and hypertonicity. There are abnormal segments of dilatation and constriction. Areas of spasm and segments of dilatation may be readily demonstrated by a roentgenogram. It is an old observation that there is a marked predisposition to colic from trivial causes in young infants, and this tendency is explained by the assumption that the stomach and intestine have not “learned” to function smoothly. Probably the myenteric plexus is immature. Curiously, the breast-fed baby presents the symptoms more often than one artificially fed.

**TREATMENT.**—The most effective treatment is an enema, about 1 ounce of plain warm water injected into the rectum with a baby syringe. Abdominal massage favors the expulsion of gas or fermented stool. A carminative may be given: 2 drops of spirit of chloroform, 10 drops of whiskey, 10 drops of the elixir of catnip and fennel, or 15 drops of peppermint water. These drugs must be well diluted with water. I am aware that carminatives are not acceptable to many prac-

tioners, who regard them as useless. Nevertheless, experience has taught that they do favor a relaxation of the intestinal spasm if not the expulsion of gas. Paregoric, in 5- to 10-drop doses diluted, may be administered when the milder drugs fail. Atropine has a remarkable antispasmodic effect in some cases. A combination of morphine ( $\frac{1}{100}$  grain) with atropine ( $\frac{1}{1000}$  grain) is the best intestinal sedative for quieting the hypermotility and spasm. The elixir of phenobarbital (10- to 15-drop doses) is being extensively prescribed for colic.

**DIAGNOSIS.**—Whenever a baby cries vigorously and persistently the possibility of *hunger* must be considered. There is no doubt that many infants showing the symptoms of colic are really hungry. After a careful palpation of the abdomen a rectal examination is required (use the little finger). Often a *rectal stenosis* due to incomplete absorption of the membrane produced by the fusion of the hindgut and proctodeum is discovered in young infants. Repeated dilatation with the finger is indicated.

*Earache* should be suspected whenever the child shows the signs of a "cold." It is a good practice to inspect the eardrums in the course of every physical examination of an infant. In older infants who suddenly manifest the symptoms of severe pain, *intussusception* should be excluded by careful abdominal and rectal palpation.

When the diagnosis of infantile colic has been made, an extensive inquiry into the *feeding* is necessary. A change in the feeding frequently cures the colic. In breast-fed babies 1 ounce of barley water before nursing seems to improve the gastro-enteric motility. In bottle-fed infants the protein in the milk should be temporarily reduced to a minimum. However, in many instances one must wait patiently for the maturity of the myenteric nervous system.

## MANAGEMENT OF ACUTE POISONINGS\*

JULIUS A. ROSSEN, M.D.†

A *poison* is any substance which through its inherent chemical properties and by its ordinary action is capable of destroying life or of seriously endangering health when it is applied to the body externally or in moderate doses internally.

*Acute poisoning* is the sum of the toxic manifestations produced by taking a large amount of a poison in a single dose, or by taking several smaller doses with such frequency as to result in sudden illness.

Two groups of patients are encountered in acute poisoning: (1) those with a definite history of the source and as often the kind of poison, and (2) those with the nature of the poison entirely unknown. We seek in the management of acute poisonings,

*First*, to determine what the poison is,

*Second*, to get rid of all or as much of the poison as possible at once,

*Third*, to administer an antidote or antagonist so as to neutralize the effects of the poison promptly,

*Fourth*, to allay symptoms and,

*Fifth*, to take all possible measures to support the patient and to ward off death.

### THE POISON

When we see the very acutely ill, and the nature of the illness is obscure, especially in children, we should never forget the possibility of poisoning. Adults often tell you of a poison accidentally or purposely taken, but the child will

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frequently pick up unknown articles, boxes, bottles, or insecticides left about the yard or house and swallow their contents. It is important however not to waste time, but to take steps to rid the patient of whatever poison he has.

### Sources of Acute Poisoning

Frequently patent medicines, insecticides, cleaning fluids, inks, shoe dyes, or hair dyes may be the source of an acute poisoning. Knowledge of the usual contents of the more common of these will be extremely helpful, if the labeled article is not at hand. There follows a list of the ingredients in preparations used by the laity:

#### A. PATENT MEDICINES

1. *Laxatives*—aloes, belladonna, calomel, phenolphthalein, saline cathartics, strychnine.
2. *Liniments*—acetone, alcohol, ammonia, capsicum, chloroform, menthol.
3. *Cold, grippe and headache cures*—antipyrine, bromides, caffeine, phenacetin, quinine, salicylates, acetanilid.
4. *Ointments*—camphor, capsicum, carbolic acid, quinine, salicylic acid, turpentine.
5. *Soothing syrups*—calomel, castor oil, opium and its derivatives.

#### B. INSECTICIDES—RAT POISONS

*Fly poison*—arsenic.

*Insect powders*—pyrethrum, sodium fluoride, sodium fluosilicate, lead, sulfur, lime, phosphorus, methyl alcohol.

*Moth poisons*—naphthalene, camphor, cedar gum.

*Rat poisons*—phosphorus, arsenic, lead, arsenous oxide, copper hydrate, lime, sulfur, nicotine, phenol, strychnine.

#### C. FIREWORKS—phosphorus, mercury thiocyanide.

#### D. FOOD.

1. *Botulism*—decomposed proteins and fats often found in cream or custards affect anterior horn cells of the spinal cord.
2. *Bacterial toxins*—Staphylococcus, Salmonella group.
3. *Poison*—such as that of the mushroom.

#### E. BITES AND STINGS

1. Snakes
2. Spiders
3. Bee stings

### Symptoms of Acute Poisoning

When a case of poisoning is encountered, the following symptoms suggest the poisons indicated:

1. *Vomiting* after a meal, *purging* and *abdominal pain*—food is suspect.
2. *Convulsions*—strychnine, nicotine, camphor, aspidium, cyanides.

3. *Coma*—opium and derivatives
4. *Rapid respiration*—atropine, carbon dioxide
5. *Delirium*—atropine group, cocaine.
6. *Dilatation of pupil*—atropine group, cocaine, nicotine.
7. *Constriction of pupil*—morphine, pilocarpine, chloral, phenol
8. *Paralysis*—cyanides, carbon monoxide, carbon dioxide.
9. *Cyanosis*—botulism, aniline, opium.
10. *Salivation*—mercury, pilocarpine
11. *Odor of breath*, sweet—hydrocyanic acid, garlic—phosphorus
12. *External tissue destruction*, yellow—nitric acid.

Symptoms of poisoning are in a great many instances so similar that it is best to divide poisons into classes and to then detail the management of each. The following classification\* seems to me the best for this purpose:

### Classes of Poisons

#### *Class 1 Gastro-intestinal irritants*

Acids

Alkalis

Irritant metallic salts

Food poisons—certain types of mushrooms (*Amanita phalloides*)

#### *Class 2 Central-nervous system irritants*

Belladonna-containing drugs

Cocaine

Scopolamine

Strychnine

Volatile oils

Food poisons—mushrooms (*Amanita muscaria*)

#### *Class 3 Nervous and circulatory system depressants*

All cardiac drugs in large doses

Coal-tar products

Cyanide

Hypnotics

Narcotic drugs

Nicotine

Most phenol-containing drugs

Food poisons (*a*) botulism (*b*) mushroom

Snake bite

### EMERGENCY TREATMENT BEFORE DIAGNOSIS IS MADE

Fortunately, in spite of a large variety of chemicals and drugs which children—and even adults—swallow, fatalities are relatively rare. The natural safeguards, vomiting and diarrhea, come to our aid, and the former, especially in the child, occurs

\* Fishbein, Morris Handbook of Therapy. American Medical Association, 535 N. Dearborn St., Chicago, Ill.

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is prolonged. There follows a comparative table quoted from Peterson, Haines, and Webster: "Legal Medicine and Toxicology."

#### VOMITING

(Frequently associated with purging and abdominal pain)

*Poisons:* Arsenic, antimony, aconite, corrosive acids and alkalis, barium, colchicum, cantharides, digitalis, copper, iodine, mercury, phosphorus, phenols, wood alcohol, veratrum, zinc, poisonous foods.

*Diseases:* Gastritis, gastro-enteritis, gastric and duodenal ulcer, cholera, cholera morbus, cholera infantum, uremia, acidosis, onset of many acute infectious diseases, the early stages of pregnancy, brain tumor.

#### CONVULSIONS

*Poisons:* Aspidium, brucine, camphor, cyanides, nicotine, santonin, strychnine.

*Diseases:* Uremia, puerperal eclampsia, tetanus, epilepsy, many acute cerebrospinal disturbances, especially meningitis.

#### COMA

*Poisons:* Opium and most of its derivatives, hydrated chloral, sulfonal, trional, veronal and other barbituric-acid derivatives, paraldehyde, chloroform, cyanides, carbon monoxide, carbon dioxide, atropine, hyoscine, the various alcohols and phenols.

*Diseases:* Uremia, puerperal eclampsia, acidosis, diabetes, cerebral hemorrhage, cerebral thrombosis and embolism, brain injury, epilepsy and other brain diseases.

#### DILATION OF PUPIL

*Poisons:* Belladonna, stramonium, hyoscyamus, scopola, and their derivatives, gelsemium, cocaine, nicotine.

*Diseases:* Certain nervous diseases causing optic atrophy, sympathetic irritation, or weakness of the oculomotor nerve.

#### CONTRACTION OF PUPIL

*Poisons:* Opium and its derivatives, physostigmine and its derivatives, pilocarpine, muscarine.

*Diseases:* Certain nervous diseases such as rabies.

#### GENERAL AND PARTIAL PARALYSIS

*Poisons:* Cyanides, carbon monoxide, carbon dioxide, botulism.

*Diseases:* Apoplexy, brain tumor, meningitis.

#### SLOW RESPIRATION

*Poisons:* Opium and its derivatives, carbon monoxide.

*Diseases:* Uremia, compression of brain, as from hemorrhage.

#### RAPID RESPIRATION

*Poisons:* Atropine group, cocaine, carbon dioxide.

*Diseases:* Acute respiratory diseases, lesions of the medulla oblongata, hysteria.

readily. Available are *three methods of emptying the stomach*: (1) the stomach tube; (2) emetics; (3) give water and produce vomiting by gagging the patient mechanically.

Usually the last of these three methods is the first one applicable to the patient. Ordinarily it is safe to tell the parents, friends or relatives over the telephone to mix the white of an egg with water, get the patient to swallow it and then produce vomiting by gagging with a spoon, or in the child even with a finger. A catheter introduced into the esophagus is more efficient. Steps must be taken to prevent aspiration.

The contents of the intestinal tract should also, in some cases, be evacuated by the use of *cathartics*. I prescribe milk of magnesia in small but repeated doses for an hour or longer.

The *standard emergency treatment* is albumen water, an emetic and milk of magnesia. A *routine antidote for unknown poison* is 2 parts of charcoal, 1 part tannic acid and 1 part magnesium oxide mixed with water and followed by lavage and purgation.

*Emetics and lavage*.—Syrup of ipecac is a safe emetic (4 teaspoonfuls), but its action is rather slow. A teaspoonful of salt or a half teaspoonful of mustard diluted with water may serve effectively. Apomorphine is too depressing, especially for children. Lavage with plain lukewarm water is indicated in infants, but in older children and adults it presents certain technical difficulties. It should be remembered that emetics are contraindicated when a half hour or more has elapsed after swallowing a corrosive poison (mercuric chloride, carbolic acid, lysol, potassium permanganate, etc.). When in doubt, lavage with a well lubricated stomach pump is safer.

In *local poisoning*, washing is employed; in *gaseous poisoning*, the patient is removed to fresh air, artificial respiration is resorted to and oxygen and carbon dioxide are given. For the emergency treatment of *bites* and *hypodermically administered poisons*, ligature, removal by suction and even excision are employed.

#### SYMPTOMATIC DIAGNOSIS

Many *diseases* and *poisons* show a great similarity in symptoms. Most diseases have a much slower onset and the course

<i>Name</i>	<i>Symptoms</i>	<i>Treatment</i>
Ammonia .....	Swelling, inflation of bronchial tubes, dyspnea.	Diluted vinegar; lemon juice; followed by olive oil; castor oil.
Antimony .....	Diarrhea soon occurs; late symptoms those of Class 3; circulatory depression.	Tannic acid, $\frac{1}{2}$ teaspoon in water; later magnesium carbonate.
Arsenic .....	Frontal headache; constriction of throat; colicky pains; eruptions on skin.	Official antidote, ferri hydroxidum cum magnesiæ oxido, 3 oz.; later castor oil.
Cantharides .....	Kidney and bladder irritation; strangury; abortion.	—
Castor oil beans ....	Collapse.	—
Chromic acid .....	Often cramps in legs.	Chalk; lime water; magnesia.
Cyanide (also Class 3) .....	May act like hydrocyanic acid; may cause stomach symptoms.	Methylene blue.
Formaldehyde solution .....	—	If swallowed, very weak solution of ammonia; diluted aromatic spirits of ammonia.
Hydrochloric acid ..	Lips and mouth show white eschar.	Lime water; magnesia.
Lead acetate .....	Colic; muscle cramps; convulsions; stupor; coma.	Dilute hydrochloric acid, teaspoonful well diluted magnesium or sodium sulfate, 30 gm. (1 oz.).
Matches .....	Phosphorus.	—
Mercury bichloride ..	Convulsions; coma; collapse.	Raw eggs and albumen water.
Nitric acid .....	Mouth and lips may be stained yellow.	—
Ovalic acid .....	Depressed circulation; cyanosis.	Magnesia; chalk; later magnesium sulfate as cathartic.
Phosphorus .....	Breath smells garlicky. Most serious symptoms sometimes slow in developing.	Use copper sulfate as emetic; wash stomach with 1:1000 potassium permanganate solution. Later magnesium sulfate.
Potassium chlorate ..	—	—
Potassium hydrate ...	—	—
Ratsbane .....	Arsenic.	—
Rough-on-rats .....	Arsenic.	—
Silver nitrate .....	—	—
Sodium hydrate ....	—	Sodium chloride (salt solution).
Sulfuric acid .....	Lips and mouth may show black eschar.	—

## DELIRIUM

*Poisons:* Atropine group, Cannabis indica, cocaine.

*Diseases:* Epilepsy, insanity, delirium tremens; organic brain diseases, e.g., meningitis; visceral diseases, e.g., nephritis.

## DYSPNEA

*Poisons:* Strychnine (in the convulsions), cyanides, carbon monoxide.

*Diseases:* Diseases of cardiac and respiratory systems, lesions of medulla oblongata and of vagus nerve.

## CYANOSIS

*Poisons:* Nitrobenzene, aniline, acetanilid, opium.

*Diseases:* Same as under dyspnea, prolonged convulsions from any cause producing cardiac dilatation.

It is essential to make a diagnosis as soon as the stomach is cleansed. Usually someone can supply definite facts as to the nature of the poison ingested. The treatment then is modified to meet the indications.

*Chemical Antidotes.*—If treatment is given promptly, the chemical antidote may be added to the water used in lavage. The more useful chemical antidotes are:

Phenol and lysol poisoning .....	10 per cent alcohol
Poisoning by heavy metals .....	6 per cent sodium thiosulfate (30 gm. to 500 cc. of water)
Acid poisoning .....	10 per cent lime water
Poisoning by alkaloids and metals ..	Tannic acid (1:1000)
Phosphorus poisoning .....	Diluted hydrogen peroxide

It is well to push the fluids, give physiological salt solution and glucose under the skin if vomiting is not allayed, and even transfusion may be indicated. The after-treatment in all cases is symptomatic. Only a few physiological antidotes are known.

## SPECIFIC TREATMENT

## Class I. Gastro-intestinal Irritants

*Immediate symptoms:* Pain, nausea, irritation or corrosion of the mouth, pharynx and esophagus, vomiting, colic and diarrhea. Collapse is produced reflexly by irritation of the vagus and is characterized by rapid heart, cold, clammy perspiration, fainting, and weak pulse with gradual failure of the heart.

*Early treatment:* Warm water, drinks containing the antidote and demulcent agents such as raw eggs, olive oil, acacia, flour and milk.

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Phosphorus .....	Breath smells garlicky. Most serious symptoms sometimes slow in developing.	Use copper sulfate as emetic; wash stomach with 1:1000 potassium permanganate solution. Later magnesium sulfate.
Potassium chlorate ..	—	—
Potassium hydrate ...	—	—
Ratsbane .....	Arsenic.	—
Rough-on-rats .....	Arsenic.	—
Silver nitrate .....	—	—
Sodium hydrate ....	—	Sodium chloride (salt solution).
Sulfuric acid .....	Lips and mouth may show black eschar.	—

## Class 2. Irritants of the Central Nervous System

*Early symptoms:* Gastro-intestinal pain often accompanied by nausea and vomiting, rapid pulse, flushed skin, and rapid respiration.

*Late symptoms:* Purging, urinary frequency, restlessness, delirium, convulsions and coma. Nephritis may be associated with the later stages.

*Treatment:* (1) Warm water drinks, (2) stomach pump, (3) emetics. Sedatives (bromides and chloral hydrate) are given by rectum. morphine for circulatory depression, chloroform for convulsions and epinephrine in circulatory failure. Keep warm.

Name	Symptoms	Treatment
Atropine .....	Flushed face; dilated pupils; dry throat; rapid heart.	Tannic acid; morphine in not too large doses.
Belladonna . . . . .	Atropine.	—
Caffeine . . . . .	—	Dry heat to body.
Camphor .....	—	—
Cannabis indica . . .	—	—
Cocaine .....	Pupils dilated; rapid heart, cyanosis.	Tannic acid if drug has entered stomach.
Hyoscyamus .. . . .	—	—
Iodoform (also Class 3) .. . . .	Nervous excitation with fever; later prostration.	—
Salicylic acid .....	—	—
Scopolamine (hyoscine) (also Class 3) . . . . .	May act like atropine but may cause circulatory depression.	—
Stramonium .. . . .	—	—

## Class 3. Nervous and Circulatory System Depressants

*Symptoms:* Depression, drowsiness, weak pulse, slow respiration, paralysis and coma.

*Treatment:* Lavage of the stomach followed by antidote if a narcotic poison—caffeine. camphor. atropine or strychnine.

Circulatory depressant—administer atropine, ergot, epinephrine or strophanthin.

Respiration fails first. Measures to be employed are:

1. Reflex stimulation of the respiratory center by use of ammonium water or smelling salts, or by the oral administration of aromatic spirits of ammonia.

2. Direct stimulation by caffeine 0.3 gm., hot coffee, strychnine 0.002 gm. or atropine 0.001 gm.

3. Artificial respiration.

For failing circulation the same methods are employed.

<i>Name</i>	<i>Symptoms</i>	<i>Treatment</i>
Acetanilid .....	Cyanosis; lowered temperature.	Oxygen inhalations; artificial respiration; sodium bicarbonate.
Alcohol (ethyl) .....	—	—
Alcohol (methyl) .....	Often late unless dose is large.	Pilocarpine hydrochloride in $\frac{1}{8}$ gr. dose; other treatment like Class 3.
Amyl nitrite .....	Respiratory and cardiac failures.	Artificial respiration; Trendelenburg position.
Aniline .....	—	—
Antipyrine .....	Profuse sweating; lowered temperature.	Sodium bicarbonate.
Bromides .....	—	—
Carbolic acid .....	—	—
Chloral .....	Pupils dilated; coma.	—
Codeine .....	—	—
Creosote .....	—	—
Digitalis .....	Cerebrum not much affected.	Tannic acid; glyceryl trinitrate hypodermically.
Ergot .....	Pupils dilated; cold surface of body; circulatory depression.	Alcohol; glyceryl trinitrate.
Gelsemium .....	—	—
Hydrocyanic acid .....	Odor of almonds; respiration and heart fail immediately.	Patient on back with feet raised; artificial respiration; ammonia inhalations; camphor and atropine injections; wash stomach.
Lobelia .....	—	—
Morphine .....	—	—
Nicotine .....	—	—
Nitroglycerin .....	—	—
Opium .....	Pupils contracted; respiration slowed.	Tannic acid; atropine; wash stomach with potassium permanganate solution 1 part to 1000.
Paraldehyde .....	—	—
Phenacetin .....	Lowered temperature.	Sodium bicarbonate.
Phenol .....	If solution strong, white eschar on lips and mouth; collapse; coma; urine black after standing.	Lavage with 10 per cent alcohol; then warm water.
Physostigma .....	Pupils contracted; may cause vomiting and purging.	—

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Morphine . . . . .	.	.
Nicotine . . . . .	.	.
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Physostigma . . . . .	Pupils contracted; may cause vomiting and purging.	.

<i>Name</i>	<i>Symptoms</i>	<i>Treatment</i>
Physostigmine .....	—	Tannic acid; atropine 0.5 to 1 mg. is physiologic antidote.
Pilocarpine .....	—	Tannic acid; atropine hypodermically.
Prussic acid .....	—	—
Resorcin .....	—	—
Salol .....	—	—
Santonin .....	—	—
Strophanthus .....	—	Glyceryl trinitrate.
Sulfonal .....	May cause prosis of eyes, suppression of urine, and papular skin eruptions.	Sodium bicarbonate; later magnesium sulfate.
Tobacco .....	Prostration.	Tannic acid if tobacco or nicotine swallowed.
Trional .....	—	—
Veratrum .....	—	—
Veronal .....	—	—

## COMMENT AND SUMMARY

In the management of acute poisoning, as in all medicine, a diagnosis is important. Just as in other fields of medicine, the history, the symptoms, the physical examination and the clinical laboratory analyses of gastric contents, urine, blood and feces must be used to the fullest extent.

The treatment requires, first, that we get rid of the poison, second, alleviate the symptoms, and third, that supportive measures be employed. Local poisons are best removed by washing. Gaseous poisons demand fresh air, oxygen, carbon dioxide and artificial respiration. Bites and stings may require removal by suction, cautery or excision. Orally taken poisons, whether they be food, minerals, alkaloids or hypnotics, necessitate prompt removal by gastric lavage, emetics and sometimes cathartics. There is no universal antidote, but the use of albumen water, milk of magnesia, or the routine antidote made up of 2 parts charcoal, 1 part tannic acid and 1 part magnesium oxide mixed with water may save many lives.

An acute illness whose nature is obscure should lead one to investigate carefully for poisons, bearing in mind that according to some authorities 80 per cent of cases of poisoning go undiagnosed. Prompt employment of the measures outlined should reduce this number greatly.

## EMERGENCY TREATMENT OF SIMPLE AND COM- POUND FRACTURES\*

J. ALBERT KEY, M.D., F.A.C.S.†

THE emergency treatment of these injuries includes the treatment of the patient at the scene of the accident, the transportation of the patient to a hospital or to some other point at which the injury will receive definitive treatment, and treatment at the hospital. The emergency treatment of any fracture includes the reduction and immobilization of the fragments in a position which will result in satisfactory function when and if union occurs. In compound fractures this also includes the treatment of the wound.

### TREATMENT AT THE SCENE OF THE ACCIDENT

This will depend upon the type of injury and also upon where the accident occurs and upon the facilities available for administering such treatment. If the patient is not very severely injured, if there are no general symptoms, if the patient has to be transported only a relatively short distance before the injury can receive definitive treatment and if this transportation can be carried out without further injuring the patient, no special treatment is necessary at the scene of the accident. However, if a bone is broken this is a surgical emergency and the patient should be transported to a hospital as soon as possible in order that treatment of the fracture may be instituted.

If the patient is seriously injured it is advisable that certain measures be taken at the scene of the accident in order to prevent further injury to the patient and prevent the develop-

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\* From the Department of Surgery, Washington University School of Medicine.

† Clinical Professor of Orthopedic Surgery, Washington University School of Medicine.

ment of shock. Other things being equal, it may be said that *the more severe the injury, the less the patient should be handled*. In other words, the severely injured patient should be left on the side of the road and made as comfortable as possible and wait there until an ambulance arrives instead of being transported some distance and placed in bed until he can be lifted again from the bed into some conveyance and taken to a hospital. This is because handling of the patient necessarily causes pain and will increase the amount of injury to the soft tissues and will tend to increase the degree of shock.

If the patient is *bleeding* and the bleeding is considerable in amount, it is very important that this bleeding be stopped as soon as possible by pressure or by an improvised tourniquet if this can be applied.

In a compound fracture it is also important that the wound be exposed and a *clean dressing* applied to the wound at the earliest possible moment. If possible this dressing should include *chemotherapy* in the wound; sulfanilamide or sulfathiazole, or preferably a mixture of the two in powdered form should be sprinkled liberally in the wound. For this reason these drugs should be made available in emergency dressing stations in industrial plants and also carried in ambulances which are sent out for the specific purpose of picking up the injured person. The placing of the chemical in the wound at this time will tend to decrease the rate of the development of infection and while it may not be necessary if the patient is going to be operated upon immediately, one can never be sure but that for some reason treatment will be delayed several hours, in which event the presence of the sulfanilamide or sulfathiazole will greatly lessen the tendency to the development of infection and will make the surgeon's work much more apt to be successful.

It is especially important that severely injured persons should be kept *warm* and *dry* and that they be given *fluids* by mouth. Likewise, unless they are suffering from brain or intra-abdominal injuries, they should be given a hypodermic of *morphine* or other opiate as soon as possible after the accident and before they are transported to the hospital.

### Splinting

The question arises as to what fractures should be splinted and how. At the present time there is a campaign on to the

Fig. 62.



Fig. 63.

Fig. 62.—Emergency splint for forearm.

Fig. 63.—Forearm splint and sling.

(From Key and Conwell, *Fractures, Dislocations and Sprains*, C. V. Mosby Co.)

effect that all fractures should be splinted where they lie before transportation is attempted. This, of course, can be carried to absurdity and much valuable time may be lost or fur-

ther injury may be done by an unskilled person who attempts to splint fractures before the patient is carried to the

Fig. 64.

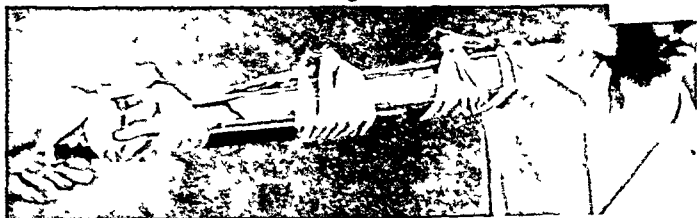


Fig. 65.



Fig. 66

Fig. 64—Emergency splint for elbow and lower arm

Fig. 65—Elbow and lower arm splint fixed to trunk

Fig. 66—Emergency dressing for injured shoulder. Pad in axilla indicated by pointer.

(From Key and Conwell, *Fractures, Dislocations and Sprains*, C V Mosby Co.)

hospital. On the other hand, patients with severe fractures who are going to be transported long distances should be

splinted before they are started on their journey. This is because splinting not only tends to lessen the pain, but also tends to prevent further damage to the soft tissues which may be incurred if the patient is transported without splintage, and tends to prevent the development of surgical shock.

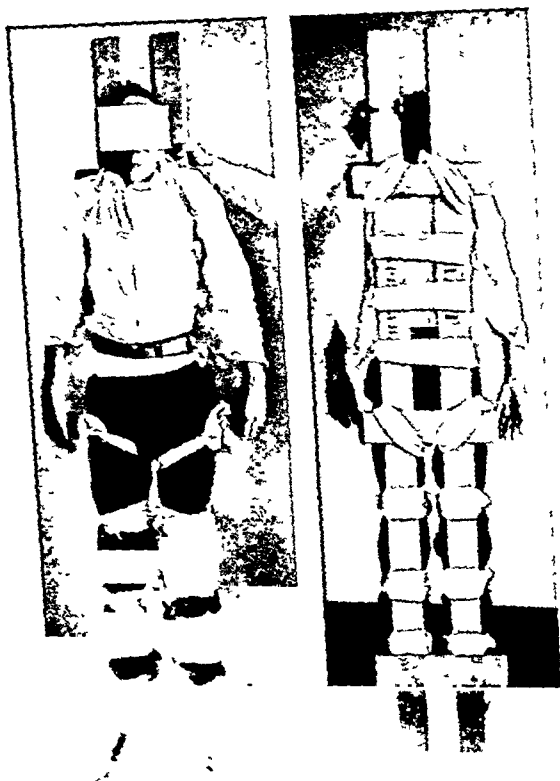


Fig. 67.—Emergency splint for spine. (From Key and Conwell, *Fractures, Dislocations and Sprains*, C. V. Mosby Co.)

Figures 62 to 70 illustrate a series of emergency splints which are suitable for both the upper and lower extremities and the spine. The splints are shown without padding and the figures are self-explanatory. These or similar splints can be improvised at the scene of the accident if splints are needed

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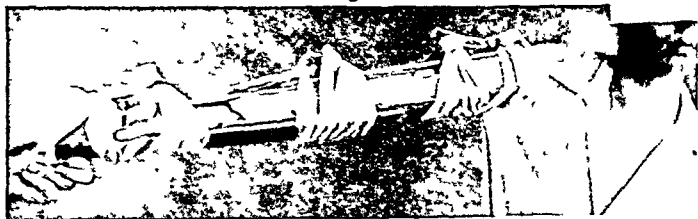


Fig. 65.

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(From Key and Conwell. *Fractures, Dislocations and Sprains*, C V Mosby Co.)

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splinted if transportation over a considerable distance is anticipated. In many instances it is sufficient merely to place the extremity on a pillow or to bind the arm to the chest or to bind the lower extremity to its fellow.

A word should be said about handling patients with *fractured spines*. There has been considerable publicity given to the statement that patients with fractured spines should be

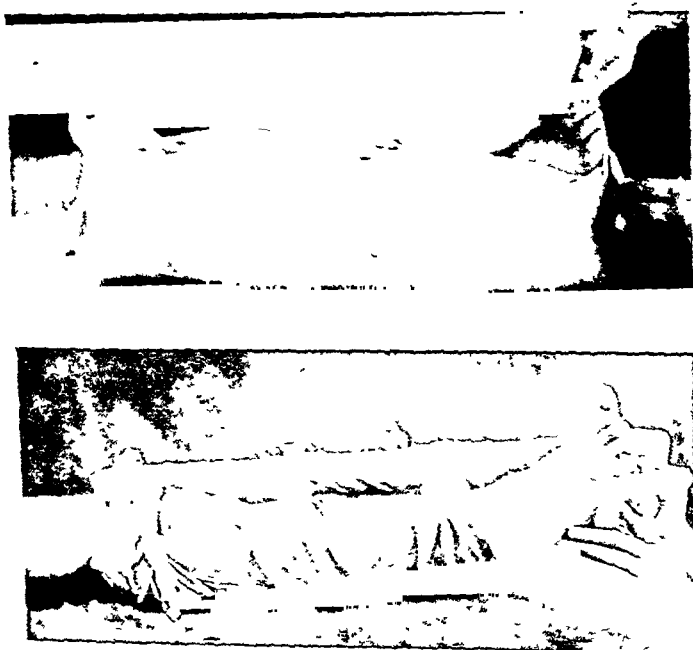


Fig. 69.—Pillow splint (above) and board splint (below) for leg and ankle fractures. (From Key and Conwell, *Fractures, Dislocations and Sprains*, C. V. Mosby Co.)

turned on their face and then picked up, because picking them up by the head and feet with the face up tends to further increase the flexion deformity and may possibly result in further damage to the spinal cord. This, I believe to be an error, because the force of the accident which has produced the compression of the spine is much greater than the small amount of pressure which may be produced by lifting the

and if commercial splints are not available (and they usually are not available). It is to be understood, however, that the application of these splints is not necessary in every case. It

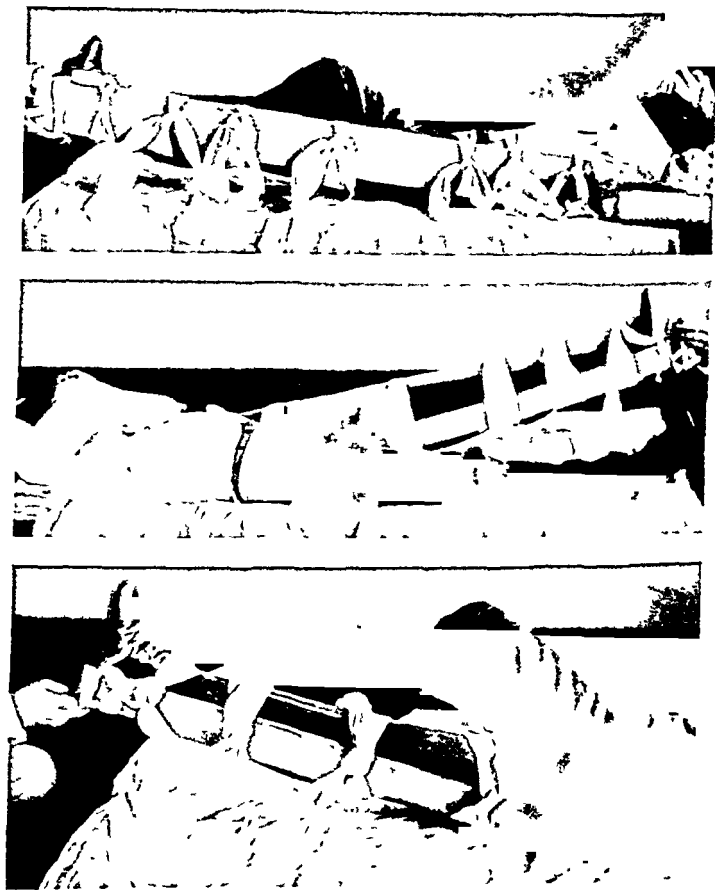


Fig. 68—Emergency splint for thigh (above) and for leg (below). (From Key and Conwell, *Fractures, Dislocations and Sprains*, C. V. Mosby Co.)

is important that a patient with a fractured femur or a fracture of both bones of the leg should be splinted if possible before he is transported for any considerable distance. Likewise, severe fractures of the upper extremity should be

splinted if transportation over a considerable distance is anticipated. In many instances it is sufficient merely to place the extremity on a pillow or to bind the arm to the chest or to bind the lower extremity to its fellow.

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*shock position* and he should be given a full dose of *morphine*. He should be given *fluids* by mouth if he can take them and usually he should be given fluids intravenously as soon as possible. If *plasma* is available a plasma transfusion can be given immediately. On the other hand, if plasma is not available 5 per cent glucose can be given intravenously (an adult receives 1000 cc.). Blood should be taken for matching and a suitable donor obtained and he should be given a transfusion of 500 cc. of *whole blood* if he does not react promptly to the above measures. If the condition is grave, *oxygen* can be administered and this will help combat the anoxemia. Fractures of the extremities should be *immobilized* with sandbags or splints, if this has not been done before the patient entered the hospital.

We have, then, as treatment of the shock patient, relief of pain by morphine, rest to the injured part, restoration of body heat by hot blankets and external heat, rest of the patient by recumbency in the shock position and isolation, the administration of fluids or blood to restore the blood volume and increase the blood pressure and, in severe cases, the administration of oxygen to combat the anoxemia.

As soon as the patient reacts from the shock, specific treatment of the fracture can be undertaken. This includes x-rays because the patient in shock should not be transported to the x-ray room before he is treated for the shock.

## 2. TREATMENT OF SIMPLE FRACTURES

When the admission, history and physical examination are completed the patient is sent to the x-ray room and roentgenograms in two planes are made of the injured parts. A definite diagnosis is now made and a decision as to the disposition of the patient is in order. That is, he may be treated as an outpatient or he may be admitted to the hospital. This will depend partly upon the nature of the injury and partly upon the physical, mental and financial condition of the patient. Regardless of the disposition, the specific treatment of the fracture should now be instituted unless it is decided that an open operation is advisable.

*Choice of Procedure.*—If the displacement of the fragments

is not sufficient to interfere with function or is so slight that an attempt at reduction may render the displacement more marked rather than improve the position of the fragments, then all that is necessary is for the surgeon to *immobilize* the fracture in a plaster-of-paris cast or splint. If displacement is present and this displacement is sufficient to interfere with function and can be corrected by manipulation or by traction or by open operation, the displacement should be corrected.

Most fractures can be reduced by closed manipulation or traction and satisfactory results can be obtained. On the other hand, in certain fractures of the patella, olecranon and single condyles of the humerus the fragments cannot be reduced and held in place by closed methods. In these not only must open operation be performed, but internal fixation of some sort must be used. Some surgeons treat most fractures of long bones by open reduction and plating or fixation with screws or other forms of internal fixation and justify their methods by the results and these are excellent in the hands of surgeons skilled in the use of this method.

By implanting one of the sulfonamide drugs in the wound, open operations for the reduction of simple fractures can be performed with less danger of infection than formerly. Consequently, I now perform open operation and use internal fixation on many fractures which I formerly treated by manipulation and immediate immobilization or by traction, because by sprinkling sulfanilamide or sulfathiazole in the wound before it is closed, the danger of infection is lessened. It is to be noted that whenever a simple fracture is operated upon it is converted into a compound fracture and the patient is exposed to the danger of infection which was not present before the operation.

*Manipulative Reduction.*—If the fracture is to be treated by manipulative reduction and immobilized in a plaster cast or splint, this should be done as soon after admission as possible, because a few hours delay may permit the development of extensive swelling which renders the treatment more difficult. Either local or general anesthesia may be used. The patient may then go home or be admitted to the hospital.

*Traction.*—Certain fractures of long bones, especially those

*shock position* and he should be given a full dose of *morphine*. He should be given *fluids* by mouth if he can take them and usually he should be given fluids intravenously as soon as possible. If *plasma* is available a plasma transfusion can be given immediately. On the other hand, if plasma is not available 5 per cent glucose can be given intravenously (an adult receives 1000 cc.). Blood should be taken for matching and a suitable donor obtained and he should be given a transfusion of 500 cc. of *whole blood* if he does not react promptly to the above measures. If the condition is grave, *oxygen* can be administered and this will help combat the anoxemia. Fractures of the extremities should be *immobilized* with sandbags or splints, if this has not been done before the patient entered the hospital.

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*Traction.*—Certain fractures of long bones, especially those

which are comminuted or oblique, are best treated by traction and if it is decided that this is the method of choice then the application of traction should be proceeded with immediately, because it is easier to prevent than to correct shortening. The surgeon should decide whether skin traction or skeletal traction is advisable and the patient should be taken directly from the x-ray room to the place where the traction is to be applied, either the fracture room or on the ward. For the application of skeletal traction it is advisable that a Kirschner wire or other means of applying traction to the bone be inserted in an operating room or plaster room where the facilities for aseptic surgery are available. The same is true of the so-called automatic splints by which the fragments are transfixed by pins and then manipulated into position under fluoroscopic control and immobilized by these same pins.

*Operative Reduction.*—If operative reduction of the fracture is decided upon there is no great hurry about proceeding with the operation and the limb may be immobilized and the operation performed the next day or after several days. On the other hand, there is no reason for delaying the treatment, because the sooner after the injury the fragments are fixed, the less hemorrhage and swelling will occur in the tissues, the more comfortable the patient will be, and it is probable that the operation can be carried out with less difficulty if it is done soon after the injury. Consequently, the skin can be prepared and the operation proceeded with immediately if the patient's general condition is satisfactory.

### 3. TREATMENT OF COMPOUND FRACTURES

If a compound fracture is present not only must the fracture be treated, but usually it is advisable to débride the wound. This means an emergency operation.

#### Diagnosis of Compound Fractures

In the receiving room it should be determined whether or not a compound fracture is present. A compound fracture is one in which communication is established between the fracture wound and the outside air. This communication may be merely a small puncture wound or a wide gaping wound, de-

pending upon the nature of the injury. It is also not infrequent for a patient to suffer a fracture of an extremity and to also suffer lacerations of the same extremity which do not communicate with the fracture and in such instances the fracture is not compound. If the wound is close to the site of the fracture and if there is an unusual amount of bleeding coming from the depth of the wound the wound probably communicates with the fracture. Also, if the blood coming from the wound can be seen to contain small droplets of fat it may be assumed that this blood is coming from the bone and that this fat is from the bone marrow. When in doubt, the laceration should be explored in the operating room and the patient treated as though the fracture were compound.

#### Which Compound Fractures Should Be Débrided?

This is a question to which the answer will vary with the individual surgeon. The fracture may be compounded *from without*—that is, by the fracturing force, in which case the overlying tissues have been crushed and more or less devitalized and foreign material including clothing and dirt may have been carried into the wound—or the fracture may have been compounded *from within*, particularly where the injury is due to indirect violence and the wound in the skin may have been produced by the end of one fragment sticking through the skin. In such instances the fracture is relatively clean unless the end of the bone has been contaminated, usually by coming in contact with the ground or pavement.

The compound fractures from within have not been subjected to radical débridement, because as a rule they will heal when reduced and treated as simple fractures. Likewise, compound fractures produced by rifle or pistol bullets traveling at high speed are usually not infected and débridement of such wounds is not necessary. On the other hand, in *war injuries* compound fractures due to shell fragments, hand grenades and fragments of bombs are usually considered to be infected, because these missiles are relatively large, irregular in shape, travel at relatively slow speed and tend to carry bits of clothing and foreign material into the tissues.

A special type of injury in civil life is a compound frac-



ing room. If he is in shock this should be treated first and he is not moved to the operating room until he begins to react from the shock. In the operating room if the patient's general condition is satisfactory he should be given a general anesthetic of pentothal sodium or an inhalation anesthetic or local anesthesia may be used, depending upon the choice of the anesthetist and the surgeon.

The patient should be placed upon the operating table without removing his emergency dressing or splint if this has been applied. If the patient is in a serious condition the general anesthetic should not be started until the operating room and team are ready, because it is advisable not to prolong the anesthesia any longer than necessary.

*Tourniquet.*—Before proceeding with the preparation of the skin or the operation a tourniquet is usually applied. This is for two reasons: (1) It conserves the blood supply of the patient. (2) It enables the surgeon to work in a relatively bloodless field and he has a better view of the damaged tissues and is able to remove foreign material more thoroughly. However, the tourniquet is removed at the end of the débridement and before the sulfonamide drugs are implanted in the wound and before an attempt is made to suture the wound, because it is important to stop all important bleeding points before the wound is sutured. In applying the tourniquet the limb should be handled as gently as possible and, if one is available, a pneumatic tourniquet should be used.

*Preparation of Operative Site.*—On the operating table the skin is prepared as for a clean surgical operation, an adequate field around the wound being cleaned and sterilized either by the dry or by the wet method. In using the *dry method* the wound is covered with a clean dry dressing and the skin is washed with benzene or ether and shaved as close to the margins of the wound as possible. The skin, not the wound, is then painted with a skin antiseptic—tincture of iodine, merthiolate, metaphen, mercresin or whatever antiseptic the surgeon prefers.

In using the *wet method* the skin wound is covered with a clean dry dressing and the surrounding skin is washed thoroughly with soap and water and shaved, the shaved area ex-

tending up as close to the wound edges as possible and covering sufficient area to allow an adequate operative field. After the limb is shaved and washed with soap and water it is then washed either with benzene or alcohol or ether and after this has dried or evaporated the skin, not the wound, is painted with whatever skin antiseptic the surgeon prefers, as mentioned above in the dry method. This preparation of the skin and of the operative field must be performed or supervised by the surgeon with sterile gloves, unless he is fortunate enough to have a well-trained assistant who can be depended upon to carry out the procedure with care and gentleness. If the wound contains dirt or grease this is washed with soap and water. The surgeon now dons a clean gown and gloves.

If a tourniquet is to be used it should be applied before the preparation of the skin is started. Having been prepared, the limb is now pulled straight, supported on sand bags or by traction and draped with sterile sheets and towels so that an adequate area is exposed for the operation.

### Technic of Débridement

The operation of débridement or mechanical cleansing of the wound is now proceeded with. The nature of this operation varies among individual surgeons. Some surgeons believe that it should include a block removal of all of the surface of the wound which is exposed and this is rarely practical. Others merely enlarge the wound and pack it open, removing any gross foreign bodies which may be found in the wound.

The following outline of the operation is one which will be successful in most cases and one which does not sacrifice any tissue unnecessarily. It is carried out in a series of steps which may be enumerated as follows:

1. *Excision of the Skin Margins.*—With toothed forceps the skin at the margin of the wound is grasped and with a knife a thin strip of skin is cut away entirely around the wound. This strip is rarely over  $\frac{1}{4}$  inch wide. It includes the skin and any subcutaneous tissue which may be adherent to the skin, but no effort is made to carry this incision into the depths of the wound. If possible the margin of the wound is excised in

one piece. However, usually the knife slips into the wound and the strip is divided at some point. When this happens the further excision of the wound margin is carried out, beginning a short distance back of where the knife slipped in so that all of that portion of the skin which is included in the margin of the wound is removed. At the same time if there is an adjacent area in which the skin is grossly devitalized by contusion so that it is perforated in small areas and ground down or crushed so that it is paper thin, this area is also excised, because if it is left it will slough and tend to cause infection. If more than one wound is present the skin of each wound is excised in succession at this stage of the operation. The knife and forceps used in this maneuver are then discarded because they are grossly contaminated, but it is not necessary for the surgeon to change his gloves as they have not touched the contaminated tissue.

2. *Enlargement of the Wound.*—The wound is enlarged by incisions up and down the extremity and the margins are retracted and its depths are inspected. The degree of enlargement will depend upon the size and depth of the wound and the damage to the underlying tissues. The longitudinal incisions are not made any longer than necessary. While it is true that wounds heal from side to side and not from end to end, it is also true that, other things being equal, small wounds heal with less complications than do large wounds. As the wound is enlarged, blood vessels which are seen or are bleeding if the tourniquet is not on, are clamped either before or immediately after they are divided, care being taken that as little blood is lost as possible. The wound should be enlarged sufficiently to permit exploration of the entire damaged area when the edges of the wound are retracted.

3. *Débridement of the Wound Proper.*—It is now possible to inspect the depth of the wound and estimate the damage to the deeper structures. The surfaces are carefully sponged. Any foreign material or foreign bodies which are seen are removed. Not infrequently the skin and fascia are stripped up and separated in layers. It is usual to excise a thin layer of the superficial fat and of the fascia which may be exposed around the margins of the wound. Likewise, severely damaged muscle

which does not contract when pinched or bleed if the tourniquet is not applied, is excised. However, no excision of undamaged muscle should be done if this can be avoided. Not infrequently masses of areolar tissue will contain small foreign bodies or dirt. It is advisable to excise such areolar tissue rather than to attempt to pick out the small bits of foreign material. If shreds of periosteum are torn off and contaminated these are excised.

At the end of this stage of the operation the wound should be relatively clean. All grossly damaged and devitalized tissue have been excised with a sharp knife. The wound should contain only living muscle and connective tissue, nerves and blood vessels, tendons and bone and periosteum, if all of these structures are exposed in the wound. During this stage of the operation it may be necessary to manipulate the extremity because not infrequently the wound extends around the bone or the bone may be entirely lifted up from its bed and foreign bodies may have lodged behind it. The entire wound should be inspected. If muscles or tendons or nerves are cut or severed and their ends are macerated or grossly contaminated, these ends should be excised with a sharp knife at this stage, care being taken to sacrifice no more of the structure than is necessary to insure the removal of devitalized or contaminated tissue.

4. *Treatment of Bone.*—In some instances the fragments of the bone are grossly contaminated—that is, the bone may have been forced out of the wound and driven into the dirt—and such bone cannot be cleaned as dirt and other foreign material have been forced into the bone spaces. This is particularly true of cancellous bone, and such areas of grossly contaminated bone should be excised with heavy bone-cutting forceps or rongeurs and no attempt should be made to scrub them clean. *Loose fragments* of bone which have been completely detached from their periosteal attachments and are devoid of circulation should, as a rule, be removed. Occasionally, they may be left for support or to prevent a loss of substance which will necessitate shortening of the extremity. However, as a rule, it is advisable to remove them, because when they are left they usually remain as inert foreign bod-

ies and even though no infection may occur they do relatively little, if any, good. This is particularly true of fragments which include the entire thickness of the cortex as such fragments show little tendency to unite and do not act like a bone graft which is firmly fixed to the bone which it is supposed to aid in uniting.

Very rarely in an *epiphyseal separation* the end of the shaft has been forced out of the wound and grossly contaminated by being driven into the dirt or forcibly coming in contact with some other foreign substance. This epiphyseal surface or end can be forcibly scrubbed with soap and water rather than excised and can then be reduced and healing may be expected to occur without deformity and without infection. The débridement is now complete. The skin edges have been excised, all visible foreign material has been removed from the wound and all grossly contaminated and devitalized tissues have been excised. In other words, the toilet of the wound is complete from the standpoint of mechanical excision.

### Irrigation of the Wound

It is now advisable to irrigate the wound with warm *normal salt solution*. This does not mean a prolonged irrigation, using several gallons of salt solution. As a rule, two quarts are sufficient. The solution is placed in the irrigating can held by an assistant and to this is attached a rubber tube without a glass nozzle. The force of the flow is controlled by raising or lowering the height of the can and by squeezing the tip of the rubber tube so that the salt solution can be squirted into all portions of the wound. This will tend to float up loose tissue, especially areolar tissue, and may bring to light and remove foreign material which may have been missed during the preceding stage. After this irrigation is complete the wound is sponged out and rendered relatively dry and is again inspected for the presence of any foreign bodies or devitalized tissue and if such are found they are removed.

### Repair of Deeper Structures

At this stage muscles, tendons and nerves which are found to be cut or torn are repaired. If torn ends have been excised

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#### Repair of Deeper Structures

At this stage muscles, tendons and nerves which are found to be cut or torn are repaired. If torn ends have been excised

during the preceding stage of the operation they are now carefully brought together and sutured with fine silk. Muscles are sutured very loosely, just enough silk being placed to hold the ends in contact. Nerves and tendons are carefully sutured with fine silk. It may be advisable in the case of both tendons and nerves to excise a small bit more from each end in order that accurate approximation of healthy tissues may be obtained.

### Reduction of the Fracture

By manipulation of the extremity or by manipulation of the ends of the fragments with bone-holding forceps the fracture is now reduced as accurately as possible. If the fracture is fairly stable no *internal fixation* is advisable. If the fracture is a long oblique fracture or a comminuted fracture it may be advisable to use some internal fixation. In certain instances of oblique fractures it may be advisable to reshape the ends of the fragments in order that they will be stable when placed in end-on contact.

In the past I have avoided the use of internal fixation wherever possible. However, during recent years with the use of vitallium and stainless steel, both of which are nonirritating, and also with the use of sulfonamide drugs which tend to lessen the tendency to the development of infection, it is now possible to use internal fixation in compound fractures with relative safety. Consequently, if plates, screws or wires are indicated to hold the fragments together, there is no reason why they should not be used and applied at this stage of the procedure, and they will render the after-treatment much more simple and tend to make it more successful. If internal fixation is used it should be applied according to good mechanical principles and plates, wires and screws should serve the purpose for which they are placed in the bone. If internal fixation is not used an assistant should hold the limb after the fracture is reduced and is stable, being careful not to move it and thus cause the fragments to be displaced while the wound is being closed.

### Final Steps in the Operation

*Final Inspection of the Wound and Control of Hemorrhage.*—If a tourniquet has been used it is now removed. The wound is carefully inspected for bleeding points of any consequence and if found they are clamped, care being taken to include no excess tissue in the clamp, and they are ligated with fine silk.

*Chemotherapy of the Wound.*—Either sulfanilamide, sulfathiazole, or a mixture of the two drugs in powder form is now sprinkled over the surface of the wound, care being taken to place some of the drug in the depth of the wound and in pockets which may have been developed in fascial spaces in order that when it goes into solution in the fluid which will collect in the wound, all parts of the wound will be exposed to a saturated solution of the drug which is used.

*Closure of the Wound.*—The question now arises as to whether or not the wound should be closed. Many surgeons believe that all compound fractures should be left open and that the limb should be immobilized and the wound permitted to heal by granulation. Others, including myself, believe that whenever possible the wound should be closed; and if the débridement has been adequate most fracture wounds in civil life can be closed if operated upon within the first eight hours and many of them can be closed within the first twelve or twenty-four hours after the injury.

In *military surgery* under war conditions most compound fractures should be débrided, the wound sprinkled liberally with sulfanilamide or sulfathiazole powder and packed open with vaseline gauze. A dry dressing should then be applied and the fracture immobilized in a plaster-of-paris cast or splint. This is because the surgeon who sutures a compound fracture should watch the patient until the danger of infection is past and under conditions of war this may not be possible.

In closing the wound no attempt is made to close it in layers as buried sutures are avoided when possible. The skin and underlying fat or superficial fascia are sutured in one layer with a continuous suture of silkworm-gut, deknatel, or

some other nonabsorbent material. Tension is avoided and if necessary tension incisions are made on either side of the wound in order to effect a satisfactory closure. Care is taken not to draw the sutures too tightly and the wound is closed without drainage. *Sulfanilamide* or *sulfathiazole* powder is then sprinkled over the wound and a dry dressing is applied. The fracture is then *immobilized in a plaster-of-paris cast* and unless evidence of infection arises this cast is not opened for inspection of the wound until sufficient time has elapsed for the wound to have healed and for the fracture to have begun to unite, usually from two to four weeks.

If, because of the lapse of too much time since the injury or for any other reason, it is deemed inadvisable to close the wound, interrupted sutures of silkworm-gut may be placed in the wound to be tied a few days later, or it may be partially closed, or it may be packed open with vaseline gauze and immobilized in a plaster-of-paris cast or splint.

The patient should be given a prophylactic dose of *tetanus antitoxin* and in severe wounds he should also be given the *combined antitoxin* for the prevention of gas gangrene.

As soon as he is able to take it after the operation, he should be given 1 gm. of *sulfathiazole* by mouth and this should be repeated at intervals of from four to six hours until the danger of infection has passed.

## OBSTETRIC EMERGENCIES ASSOCIATED WITH HEMORRHAGE\*

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HEMORRHAGE in the gestational state may occur antepartum, intrapartum, or postpartum. Discussion of "hemorrhage" in the various stages of pregnancy will include the problems associated with threatened and incomplete abortion, premature separation of the normally implanted placenta, placenta praevia, rupture of the uterus, injuries to the birth canal and postpartum hemorrhage.

Although a bloody discharge may or may not be of importance, the consideration of *any* abnormal bloody flow as an obstetric emergency is deemed advisable. This attitude is taken in the conviction that the successful maintenance of a product of conception may depend on the prompt attention of the physician to this symptom.

Considering *antepartum bleeding*, one recognizes that some women may have a flow at the time of an expected menstruation even after pregnancy has been diagnosed. In addition, there are various cervical lesions which may bleed during this period. These include erosions, polyps and even malignancies of the cervix which are usually readily recognized on speculum examination and can be treated accordingly. However, hemorrhage which occurs in the first trimester of pregnancy is always suggestive of threatened abortion and should be treated as such after eliminating the above cervical causes. There seems to be some confusion in the technical application of the term *abortion*. In this discussion abortion will be used to indicate the termination of pregnancy before the sixteenth week; *miscarriage* will be reserved for termination be-

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tween the sixteenth and twenty-eighth week, and *premature labor* will be reserved for termination between the twenty-eighth week of gestation and term.

### ABORTION

Hemorrhage is one of the serious symptoms of abortion. An abortion may be threatened or inevitable; it may be complete or incomplete; it may be habitual.

*Etiology.*—As will be developed later, it is not necessary to delve into an exhaustive review of the etiologic factors involved in abortion. One of the most frequent causes is the death of the fetus. This death may be due to an abnormal development, which may be maternal, fetal or even paternal in origin. This type of pregnancy will go on to abortion or miscarriage and becomes an emergency only if it develops into an incomplete abortion with hemorrhage as a complicating factor. This will be discussed later under the heading of incomplete abortion.

Endocrine or vitamin deficiencies or dysfunctions must also be seriously considered as factors in threatened interruptions of pregnancy. Threatened abortion constitutes an obstetrical emergency because it can be helped materially by prompt application of certain measures which will be discussed later.

The principal element of abortion which presents as an emergency is *hemorrhage*, frequently associated with pain or cramps in the lower abdomen. Successful prevention of abortion revolves around the speed with which the patient is given adequate care.

Habitual abortion does not constitute an obstetrical emergency and will not be discussed at this time.

#### Treatment of Incomplete Abortion

When abortion has progressed to the point where an obstetrical emergency exists because of hemorrhage, *the uterus should be emptied* by conservative methods at once. The choice of procedure depends on the consistency and patency of the cervix. Usually part of the product of conception has been expelled in the early weeks of pregnancy and hemor-

rhage may occur from remnants of chorionic tissue which are still attached to the uterus and which require operative removal. In the latter weeks of the first trimester, after the placenta has formed, the abortion is often incomplete as a result of partial placental separation.

A mild semimarcosis is induced, usually with one injection consisting of morphine, grain  $\frac{1}{8}$ , and scopolamine, grain  $\frac{1}{150}$ . The patient is placed in a lithotomy position, the genital hair is shaved, an external scrub of soap and water is given and the parts are rinsed with a mild antiseptic such as 1:1000 bichloride of mercury. The patient is draped with sterile sheets.

If possible, subsequent manipulation should be performed without aid of anesthesia, in order to diminish the danger of perforating the very soft uterus. With the patient in a mild state of semimarcosis, manipulation must be performed in a most gentle manner to avoid causing unusual pain or distress. If anesthesia is necessary, nitrous oxide-oxygen is employed.

It is usually considered best not to catheterize these patients since the threatened abortion has frequently been present for several days before becoming inevitable and infection is likely to be present.

The patient is carefully examined bimanually. Frequently remnants of the product of conception will be found to lie in the cervical canal. When the size of the uterus and the consistency and extent of dilatation of the cervix have been determined, effort can be made to empty the uterus.

If the cervix is found to be sufficiently dilated, the interior of the uterus is explored digitally and remnants of tissue are loosened. An antiseptic solution such as 1 per cent acriflavine in glycerin is used liberally for lubrication and antisepsis. Having made such preliminary exploration, a bivalve speculum is inserted and the anterior cervix is grasped with a sponge holder. The vagina is wiped with acriflavine-glycerin solution and the uterus is explored with a sponge holder to remove loose fragments of tissue. The uterine walls may also be wiped with a rounded instrument to detach and remove such fragments. At this time an ampule containing 0.02 mg. of ergonovine is given intramuscularly. All intra-uterine manipulation must be done with extreme care and a most gentle touch.

Some observers employ a warm 1:1000 potassium permanganate *intra-uterine douche* under low pressure at 110° F., using a Bozeman nozzle, upon completion of the emptying of the uterus. This procedure is performed for the purpose of rinsing out fragments of tissue, washing out the cavity of the uterus and aiding, by heat, in the contraction of the uterus. The procedure has been subject to considerable criticism, but has proved eminently satisfactory on the service of the St. Louis Maternity and St. Louis City hospitals.

Should the cervix not be sufficiently dilated to allow for the emptying of the uterus, *dilation* may be effected by means of Hegar or Goodell dilators. However, such mechanical dilation is extremely dangerous when performed upon these soft and friable tissues.

If the cervix is too rigid for dilation, the canal should be packed tightly with 1-inch gauze saturated with 1 per cent acriflavine in glycerin. When the pack is removed at the end of twenty-four hours, the products of conception usually come away with it; if they do not, the procedures already discussed can be carried out.

As soon as the uterus has been emptied, it will contract vigorously and hemorrhage will cease.

### Emergency Treatment in Threatened Abortion

In the past the treatment of these cases included bed rest, heavy sedation including opiates, elevation of the foot of the bed, ice bags to the lower abdomen and other symptomatic measures. In the light of present-day information, various other factors, such as endocrine and vitamin activity, must be considered in any therapeutic regimen. The corpus luteum and its hormones are recognized as of vital importance in the development of the human ovum and in the physiology of implantation and development of the fertilized ovum, and the clinical use of *corpus luteum extracts* and *progesterone* is of unquestioned value in the treatment of threatened abortion.

The following plan of treatment based upon the use of *progesterone* and its related compound, *anhydro-hydroxyprogesterone*, has been successfully employed in our practice. At the first sign of bleeding, the patient is put to bed and

5 or 10 mg. of progesterone are administered hypodermically. A very mild sedative such as sodium bromide or small doses of barbiturates may be given if the patient's nervous state warrants. At the same time, anhydro-hydroxy-progesterone is started orally. The dosage varies from 30 to 100 mg. per day depending on the availability of the drug and the degree of bleeding or pain. It averages 80 to 60 mg. per day. We have administered daily as much as 30 mg. of progesterone hypodermically and 100 mg. of anhydro-hydroxy-progesterone orally. The hypodermic therapy is discontinued after one or two injections and we continue with the oral form. After two or three days the oral dosage is reduced to 40 to 30 mg. per day. Therapy is continued for at least three or four days after all evidence of bleeding has disappeared. Inasmuch as threatened abortions usually occur at the time of an expected menstrual period, treatment is frequently resumed at the time of the next two or three such critical periods. Under such a regimen, in which the treatment has been started promptly and dosage has been adequate, we have maintained 80 per cent of threatened interruptions of early pregnancy associated with bleeding.

Thyroid therapy and vitamin therapy are valuable in habitual abortion, less so in the emergency care of threatened abortion.

It is advised not to use morphine or opiates in threatened abortion. Many observers feel that opiates produce an unusual degree of relaxation of the cervix which tends to increase the possibility of a threatened abortion becoming inevitable.

#### PREMATURE SEPARATION OF THE NORMALLY IMPLANTED PLACENTA

Premature separation of the normally implanted placenta (known also as "ablatio placentae" and "abruptio placentae") is an acute obstetric emergency associated with antepartum hemorrhage. The placenta may be completely or only partially separated from its implantation site and the gravity of the accident varies according to the extent of separation and degree of hemorrhage. The effused blood may be entirely retained within the uterine cavity (concealed accidental hem-

orrhage) or the blood may escape externally through the vagina (external accidental hemorrhage).

*Incidence.*—If we include all cases in which there is a separation of the lower pole of the placenta, particularly if the placenta site is abnormally low, the condition occurs as frequently as once in 200 pregnancies. The more severe form of this accidental hemorrhage is noted about once in 500 to 1000 pregnancies.

*Pathology.*—Premature separation of the placenta is initiated by an escape of blood into the decidua basalis. In its earliest stages, the condition consists usually of a decidual hematoma which leads to separation and ultimate elimination of function of that part of the placenta adjacent to the hematoma. In early stages, the diagnosis is made only upon examination of the freshly delivered placenta which presents a dark area of clotted blood at the site of the decidual hematoma. When the decidual hematoma is more extensive, the area of separation is more widespread and extends to the margin of the placenta. The escaping blood makes its way between the membranes of the uterine wall and eventually appears externally.

"Concealed" accidental hemorrhage thus is liable to occur when the blood lies behind the placenta and the margins of the placenta remains adherent; when the placenta is separated completely and the membranes retain their attachment to the uterine wall; when the blood gains access to the amniotic cavity after breaking through the membranes; and when the presenting part is so tightly applied to the lower uterine segment that the blood cannot escape externally.

In most cases the membranes are gradually dissected from the uterine wall and blood eventually escapes through the cervix and from the vagina. The seriousness of the situation depends upon which portion of the placenta becomes separated first and how much hemorrhage has occurred.

In the most severe cases, "uteroplacental apoplexy" occurs. The blood, which is under pressure, makes its way between the muscle bundles of the uterus. Sometimes the entire thickness of the uterine wall is filled with extravasated blood until hemorrhage is noted under the serosa. Under such circumstances, the uterus presents a mottled appearance, is not contractile, and is doughy to leathery in consistency.

*Etiology.*—As is noted so frequently where the primary cause of a condition is understood imperfectly, many conditions have been cited as etiologic factors in premature separation of the normal implantation placenta. Trauma, shock, anomaly of the umbilical cord, endometritis, nephritis, toxemia, torsion of the uterus, associated acute infections, specific toxins such as histamine, hydramnion and endocrine deficiency have all been held responsible for this condition.

*Symptoms and Diagnosis.*—The symptoms of this acute obstetrical emergency are *hemorrhage, abdominal pain, shock and gradual enlargement of the uterus* which accompanies a concealed hemorrhage. The most critical symptoms occur in those cases of concealed hemorrhage. Intense colicky pain is noted, and the uterus, as it becomes progressively larger, presents on palpation a boardlike consistency with elimination of the alternate contraction and relaxation usually noted. In such extreme cases, the child is dead and no fetal heart beat is elicited.

When hemorrhage is external, the pain is frequently less severe, the uterus enlarges slightly if at all and its consistency may not be altered.

#### Treatment

Premature separation of the normally implanted placenta is an obstetric emergency because the life of the mother is endangered. In the more serious forms, the life of the mother can be saved only by emptying the uterus promptly. When the separation is partial and the loss of blood is slight, expectant treatment may be followed, labor may be allowed to take its natural course and interference may be indicated only if the symptoms become urgent.

In the more severe cases, *abdominal section* (hysterotomy) should be performed at once and the uterus retained or removed according to whether it contracts satisfactorily upon being emptied. This procedure is indicated whether labor has set in or not. The use of bags to dilate the cervix from below is rarely indicated on account of their slow action.

*Blood transfusions* should be available as soon as the condi-

tion is diagnosed and should be given freely depending on the degree of shock.

When delivery is effected through the natural passages, it is not possible to evaluate the degree of intramuscular hemorrhage and the patient must be observed carefully for post-partum hemorrhage which occurs frequently if the tonicity of the uterus has been impaired by intramuscular hemorrhage. Necessary material for emergency packing of the uterus, blood transfusion and an operating room in readiness should all be available.

#### PLACENTA PRAEVIA

The most common cause of hemorrhage in the latter third of pregnancy is placenta praevia—that condition in which the placenta is implanted in the lower uterine segment and either partially or completely covers the internal os of the cervix. Hemorrhage in placenta praevia results from the partial separation of this placenta.

*Placenta praevia centralis* is that condition in which the internal os is completely covered by placental tissue. It is not necessary that the center of the placenta lie over the os. *Placenta praevia partialis* exists when the placenta covers a portion of the internal os, but a separation which is free from placental attachment can be detected by the examining finger. *Placenta praevia marginalis* occurs when the lower edge of the placenta just approximates the internal os.

The clinical determination of the variety of placenta praevia depends on the condition of the cervix. With a tightly closed cervix which barely admits one examining finger or a cervix which is only slightly open, the actual extent of the placenta praevia may be indeterminate. The handling of such a case is dependent therefore to a great extent on the amount of hemorrhage which constitutes the obstetric emergency rather than on the anatomical variety of placenta praevia.

*Frequency.*—Placenta praevia is a fairly rare complication with a considerable statistical variation in frequency. In general, it occurs approximately once in 700 to 1000 pregnancies in private practice.

*Etiology.*—Placenta praevia occurs more frequently in multiparae. Careful history frequently reveals existence of a pre-

vious abortion, some infectious process resulting in endometritis or subinvolution which indicates an abnormality of the endometrium. Thus, if a poor endometrium is available and no well-marked folding of the endometrium is present in the upper aspect of the uterus, the fertilized ovum may drop low in the cavity before it embeds for its nidation.

Another theory as to the development of placenta praevia originates in the belief that part of the chorion laeve continues to grow instead of undergoing involution early in pregnancy, with the result that part of the placenta comes in contact with the decidua capsularis. This placenta reflexa gradually bridges over the internal os and eventually comes in contact with the decidua vera to which it fuses, forming in effect an extension of the decidua basalis. The villi on the upper aspect of the reflexa atrophy, leaving a large broad placenta covering the lower uterine segment and occluding the internal os.

*Symptoms.*—Painless bleeding during the latter third of pregnancy is the cardinal symptom of placenta praevia. Frequently it occurs without warning in an individual who has had a previous normal pregnancy. The initial bleeding is rarely sufficient to prove fatal, and ceases spontaneously to recur again without warning. In some cases the bleeding does not cease entirely and there is an ensuing continuous discharge of small quantities of blood.

*Diagnosis.*—Placenta praevia is diagnosed by *vaginal examination* which reveals its presence.

If the hemorrhage is not great and the patient can stand such manipulation, it may be possible to disclose the placenta praevia by *roentgenologic examination*. An opaque medium such as 12.5 per cent sodium iodide is instilled into the previously emptied bladder and an anteroposterior x-ray film is made. In the normal situation, the fetal head can be found close to the bladder margin as outlined by the opaque substance. If placenta praevia is present, the concave mass of the placenta will be found to form a distinct gap between the head of the bladder margin. Recently with "soft tissue" x-ray technic, the placenta has been visualized without employing opaque media.

## Treatment

The immediate treatment of placenta praevia is the treatment of excessive blood loss and shock. *Morphine*, absolute *quiet in bed*, *elevation of the foot of the bed*, and *heat* in the form of hot water bottles and blankets are important. As soon as the patient has reacted to these measures and has shown improvement from the internal hemorrhage, she should be taken to a hospital, being transported in a reclining position.

*Blood transfusions* in amounts of 500 to 1000 cc. are all-important. Vaginal examination to confirm a proper diagnosis must be reserved until everything is in preparation for aseptic examination and preparations have been made for operative intervention if necessary.

The management of placenta praevia may depend on various factors. If the hemorrhage occurs *before the stage of fetal viability*, treatment may be expectant if the hemorrhage is not too severe and does not recur to a great extent after the initial episode. After combating the initial loss of blood, no further steps need to be taken until the stage of viability is reached, except the enforcement of absolute bed rest. Frequently such a temporizing attitude is taken without an established final diagnosis but always with the complete understanding of the patient as to the potential gravity of the situation.

*When the fetus is viable*, particularly past the thirty-second week of pregnancy, unless the degree of bleeding is trifling, pregnancy should be terminated in the most convenient manner as soon as the patient is in proper condition. Again here, the time at which such termination is effected will depend on the degree of hemorrhage and the duration of pregnancy. When the patient is in proper condition and blood transfusions are available, she should be examined for diagnosis. The type of placenta praevia and the condition of the cervix should be ascertained. It is the belief of the author that in all cases of *central placenta praevia*, excepting the occasional one in multiparae with a considerably dilated cervix, pregnancy should be terminated by cesarean section.

*Partial placenta praevia* in primiparae is usually best treated by cesarean section, but in multiparae its treatment will depend on the condition of the cervix. Should the cervix be

long and firm, cesarean section is the procedure of choice. If the cervix is soft and dilatable, the membranes may be ruptured and either a large bag may be inserted intraovular or packing may be employed if the operator understands the proper technic of packing.

*Marginal placenta praevia* is usually ably handled by simple rupture of the membranes which allows the presenting part to tampon the separated placental margin against the cervix, thus controlling the bleeding. In any form of treatment which employs delivery from below, version at the time of complete dilatation of the cervix is indicated.

Manual dilation of the cervix is never employed. Whenever cesarean section is employed, the uterus should be packed tightly because the bleeding has occurred from the non-contractile portion of the uterus.

The importance of frequent blood transfusion cannot be overemphasized.

#### INTRAPARTUM HEMORRHAGE

Intrapartum hemorrhage includes all bleeding from any cause occurring during labor. Such intrapartum bleeding may be due to placenta praevia or premature separation of the normally implanted placenta as discussed previously. Other important causes are injuries from attempts at operative delivery and rupture of the uterus. Bleeding during labor may occur from ruptured varices, tumor growths or spontaneous laceration of the cervix or vaginal floor.

##### Rupture of the Uterus

Rupture of the uterus *during labor* is very serious. Hemorrhage is a feature. Unfortunately the external bleeding may be slight but the concealed hemorrhage may be so extensive that, unless an early diagnosis is made, death frequently ensues.

*Spontaneous rupture before labor* occurs infrequently and is due either to an inherent defect in the musculature or to an acquired defect such as a poor cesarean scar or a previous operative scar which may cause a thinning of the uterine wall.

If labor has started, a frequent factor in rupture of the

uterus is *dystocia*. This may be due to contracted pelvis, oversized fetal head or abnormal presentation as well as obstruction from any other cause such as tumors.

*Traumatic* rupture of the uterus may occur from introduction of bougies, craniotomy instruments or even forceps blades. Traumatic rupture of the uterus may also follow attempts at manual dilation of the cervix during labor.

*Symptoms*.—Rupture of the uterus is usually accompanied by *pain* and *bleeding*. The signs usually point to an acute abdominal condition which usually requires laparotomy even though the absolute diagnosis may not seem clear.

When labor has once begun, the sharp pain may be followed by cessation of labor contractions and hemorrhage. The degree of collapse and shock depends on the degree of hemorrhage.

*Treatment*.—Most instances of uterine rupture are avoidable. When previous operations have been performed on the uterus, the patient should be observed closely during the last few weeks of pregnancy. If *cesarean section* is deemed necessary, it should be done electively or after a rigidly observed trial of labor.

Attempts at operative delivery requiring intra-uterine manipulations must be carried out with great care. Version for transverse presentation is an extremely hazardous procedure. When intra-uterine manipulation is necessary, complete relaxation of the uterine musculature by use of complete surgical anesthesia is advocated.

Rupture of the uterus at any time requires immediate *laparotomy* with either repair of the tear or removal of the uterus. *Blood transfusion* must precede any operation and must be employed frequently during and after such procedures.

#### POSTPARTUM HEMORRHAGE

Postpartum hemorrhage may be defined as excessive bleeding from the genital tract which occurs between the time of the birth of the child and the end of the puerperium. Such a state is considered an obstetric emergency because the patient may become exsanguinated within a very few minutes.

Clinically, a loss of more than 600 cc. of blood may be re-

garded as constituting a hemorrhage. When the loss approaches two or more liters the situation may jeopardize life. The hemorrhage may take the form of a continued dribble or ooze of blood which continues after expulsion of the placenta, or the loss may occur very rapidly, the blood coming in continuous or intermittent gushes. In either case the total blood volume is gradually or rapidly reduced to a point which can produce profound shock and the patient is in imminent peril of her life. The possibility of concealed hemorrhage is sometimes overlooked until the systemic evidences of anemia and impending shock call attention to its existence. Palpation of the fundus shows a soft distended uterus sometimes extending upward beyond the umbilicus. Massage of this dilated uterus will release a great quantity of free blood and clots.

*Etiology.*—Serious bleeding following the birth of the child is usually due to one of three causes:

1. *Atony.* Atony of the uterus may be due to primary uterine inertia or a secondarily acquired inertia as noted in exhaustive labors. The latter may frequently follow the so-called "forced labor" initiated, continued and completed with the aid of pituitary products. Postpartum atony may also follow overdistention of the uterus, seen in cases of multiple pregnancy and hydramnion. Following any of these conditions, the uterine musculature may remain flabby and atonic, fail to respond to ordinary measures and lead to postpartum hemorrhage.

2. *Retention of partially separated placenta or individual cotyledons.* As long as the placenta remains completely attached to the uterine wall, there can be no possibility of hemorrhage. Once it has become partially separated, hemorrhage may occur from the torn vessels at that site. This incomplete separation of the placenta may be due to an abnormal attachment of the placenta, to improper management of the third stage of labor, or to the retention of cotyledons.

3. *Deep tears involving the tissues of the birth canal.* These include particularly cervical lacerations and lacerations of the vaginal walls, but occasionally serious tears involve the vulva itself. Cervical tears may be very extensive and even involve large divisions of the uterine arteries, in which case severe blood loss may occur quite rapidly. Vaginal tears usually do not cause extensive bleeding but can do so if large vessels are severed. This

is particularly true if the tear extends laterally and involves the pampiniform plexus or extends into large varicosities of the vagina. Vulvar tears likewise only occasionally cause marked bleeding. Deep tears may follow spontaneous delivery but are more often encountered following forceps delivery. Forceps are of immense value when used properly and particularly when used at the proper time but forceps are likewise dangerous and can cause great injury. This is particularly true when forceps are applied prematurely and forceful delivery is done or when forceps are used by the unskilled operator.

### Prophylaxis

Of the three causes listed, the second is the most usual in the production of serious bleeding. Most often this can be obviated by the *proper management of the third stage of labor*. The incidence of abnormal placentae with succenturiate lobes or other forms of abnormal attachment is small. The third stage of labor is a physiologic process whereby the placenta is separated and expelled from the uterus by a series of rhythmic contractions. In most instances this can be accomplished uneventfully without interference. However, at times there occurs a continuously enlarging retroplacental hematoma, the proportions of which may result in an excessive blood loss if it is allowed to continue. For this reason and to facilitate expulsion we have instituted the practice of assisting in the third stage.

Our regimen is quite specific. First, no oxytocics are given prior to expulsion of the placenta. It is true that only rarely does retention of the separated placenta occur subsequent to cervical contraction induced by oxytocics, but even the occasional one is sufficiently troublesome to warrant waiting until the third stage is completed before using these drugs. Secondly, no type of manipulation is performed until the placenta is definitely separated unless persistent bleeding renders this imperative. Separation is readily recognized by the four signs of Ahlfeld. Usually someone places a hand on the fundus in order to ascertain the onset of contractions and the rising of the fundus and to warn the operator if the uterus is being distended by retroplacental blood. When separation is completed, then expulsion is completed by the Credé method.

One cubic centimeter of ergonovine is given intramuscularly and the uterus is gently kneaded to maintain contractions of the uterus until the drug has become effective. Additional ergonovine may be given intravenously if necessary. Inspection of the placenta then follows to be certain no break in the continuity of blood vessels of the membranes is present (succenturiata, duplex, etc.) and also to look for missing cotyledons. All patients are carefully observed by the intern for an additional hour to be certain no signs of abnormal bleeding occur.

Too often the obstetrician is anxious to complete the third stage immediately and often only ends up with a serious case of bleeding which prolongs instead of shortens the time spent in the delivery room. When vigorous attempts at expulsion are made before complete separation occurs, partial separation usually follows. Bleeding of varying magnitude ensues before separation and expulsion can be accomplished by the Credé procedure and the condition of the patient may suffer seriously. Retained cotyledons and inversion of the uterus are other sequelae.

#### Treatment

The treatment of postpartum hemorrhage varies according to the cause. *If the uterus is well contracted*, lacerations should be sought for by exposure of the parts with a speculum; bleeding vessels if found are ligated and lacerations are repaired, whereupon the bleeding will cease at once.

*If the uterus is soft and relaxed* after the placenta has escaped, as in atony and in retention of the partially separated placenta or of individual cotyledons, it is stimulated to contraction by *pressure* and *massage*. One hand inverts the lax abdominal wall behind the uterus, the other makes pressure just above the symphysis and the uterus is firmly squeezed between the two, and at the same time is pulled upward, thus making anteroposterior pressure and putting the uterine arteries on the stretch. If the placenta remains attached, with free bleeding, the Credé mode of expression is utilized and if this is unsuccessful manual removal under deep anesthesia is done. Retained cotyledons and succenturiate lobes are likewise re-

moved manually. Rarely placenta accreta may exist, in which case hemorrhage is usually slight and time may be given before manual removal is attempted. Of course, in accreta hysterectomy must follow.

Most really dangerous postpartum hemorrhages occur after expulsion of the placenta and if simple massage or oxytocics do not promptly control the bleeding, the uterus is packed firmly with sterile gauze dipped in 1 per cent neutral acriflavine. The vagina is packed with a second strip of gauze. If the bleeding still continues, the situation is grave indeed and heroic measures are required. The cervix may be sewn shut over the packing to retain the blood in the vagina in the hope that clotting will occur. Long Pean clamps or a ligature may be placed on the uterine arteries through the vagina or the arteries may be exposed vaginally and ligated. Finally abdominal hysterectomy may be necessary. These are desperate measures for desperate conditions, and if they are required, the prognosis is not promising. Compression of the aorta through the abdominal wall is usually not satisfactory.

While local efforts at combating the hemorrhage are in progress, *systemic measures* to combat the shock and anemia should be inaugurated. The failing heart is temporarily stimulated with caffeine benzoate or coramine. Most important is blood transfusion, 500 cc. or more, or the use of blood plasma when available. In the meantime temporary replacement of blood volume by intravenous fluids or gum accacia is indicated, as well as general methods of combating shock. The value of repeated transfusions cannot be stressed too much.

Delayed postpartum hemorrhage occasionally occurs, and is usually due to overlooked retention of cotyledons; however, a retained organized clot may be responsible, as well as a few other rare conditions. Treatment depends upon the etiology.

## EMERGENCIES ARISING FROM PHYSICAL AGENTS\*

A. J. KOTKIS, M.D.†

THE present day tremendous step-up in industrial production and increases in speed and modes of travel, as well as the increased use of inflammables, bring the average human into contact with a wide variety of agents—physical and chemical. Undue contact or exposure to any one or a combination of these agents may give rise to an emergency, the degree or severity of which will depend upon the type of agent, the intensity of the energy, and the duration of the contact.

In the majority of emergencies the principal result is *shock*, or what some medical investigators call *peripheral circulatory failure*. This constitutional phenomenon of shock is divided into two stages: first, primary or initial shock, and second, secondary or late shock. The prognosis in any emergency will depend upon three factors: (1) on how effectively the primary shock can be relieved, (2) on how early and effectively steps were taken to prevent or combat secondary shock, and (3) on how early and effectively the vital requirements (supportive therapy) are maintained. The best methods, however, for treating these emergencies arising from physical agents are *intensive safety campaigns*.

### BURNS

*Classification.*—Burns are divided into five classes according to the degree of tissue destruction. These may be listed briefly as follows:

1. *First degree burns* are caused by a temperature of about 140° F. and involve only the superficial layers of the epidermis,

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and are characterized by erythema, slight edema, smarting, and tenderness.

2. *Second degree burns* are caused by temperatures between 160° F. to 210° F. and involve the whole thickness of the epidermis as far down as the capillary layer of the corium, which layer is usually left intact. These burns are characterized by the formation of vesicles, more marked inflammation, edema and considerable pain.

3. *Third degree burns* are caused by temperatures greater than 210° F. and involve not only the epidermis but the upper layers of the corium.

4. *Fourth degree burns* are caused by a more prolonged exposure to a temperature similar to that responsible for burns of the third degree, or to a higher temperature. They are characterized by complete destruction of the skin and subcutaneous areas, and extend to the muscles.

5. *Fifth degree burns* may extend down as far as the bone and are characterized by a carbonization of the parts involved.

#### Factors in the Making of Emergencies in Burn Accidents

1. The initial factor is the *burn* by its extent in depth and surface area, as well as location. It becomes the principal factor only when it is so grave that death results immediately or within twelve hours.

2. The second factor, and most frequently the principal factor, is the *secondary shock* (peripheral vascular failure) occurring within twenty to forty hours, usually resulting in death if the latest approved constitutional therapeutic measures are not instituted early. It has been shown by various investigators that this vascular failure is due to stasis and increased permeability of the capillaries, producing a pronounced loss of plasma proteins from the blood stream to the tissue spaces with a consequent increase of red cells and hemoglobin in the blood. These blood changes start about twenty-four hours after the accident. Experimental evidence has shown that a burn occupying about one sixth of the body surface may cause a loss of blood plasma proteins up to 70 per cent of blood volume within twenty-four hours. In view of these facts, it is imperative that *plasma transfusions* and not *blood transfusions* be administered at the earliest possible moment—quantities from 2000 to 5000 cc. have been ad-

ministered within forty-eight hours. Such measures as bed rest, morphine for pain, maintenance of body heat by blankets and light cradles, débridement of dead burned tissue, the aseptic opening of blisters, and topical applications of astringent and antiseptic solutions to the burned areas are very important but essentially contributory efforts.

3. The third factor is *toxemia* occurring after the secondary shock is fairly well under control. This toxemia is the result of the absorption of other tissue toxins and also bacterial toxins from secondary infection. The discussion and treatment of this condition belong in the late treatment of burns, although measures to minimize it should be taken in the emergency treatment.

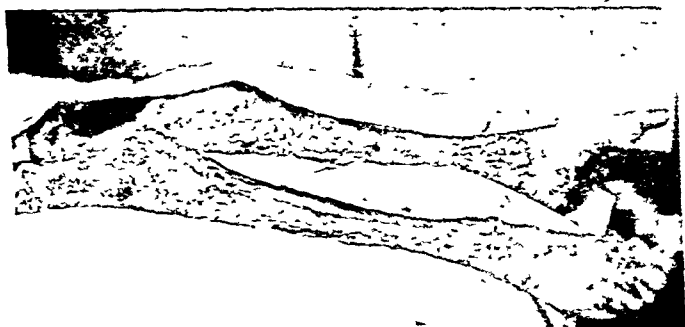


Fig. 71.—Gasoline burn accident resulting in third and fourth degree burns of lower extremities and second degree burns on hands and lower third of forearm (approximately 50 per cent of area). Removed to Firmin Desloge Hospital (180 miles) twenty days following accident. Patient received in condition of great toxemia from massive purulent exudates and sloughing tissues. Plasma and blood transfusions. Continuous flow bath with force spray to loosen sloughs and purulent exudates assisted in débridement and prepared for skin transplanting. Hospital stay 100 days—discharged walking on crutches.

### Signs and Symptoms of an Emergency

In *grave* emergencies the patient is usually in a coma or stupor, although sometimes he or she may be conscious and rational. The systolic blood pressure is usually below 100 mm. The pulse is very rapid and the respiration may reach 35 to 40 per minute. Rectal temperature starts rising immediately and may reach 104° F. within twelve hours. Pain is a vari-

able symptom; the semilethargic patient may be numb to pain but the majority of the conscious, rational patients invariably complain of severe pain.

In *severe* emergencies where there is a possibility that the patient may die within thirty-six to forty-eight hours, the signs and symptoms present within the first twenty-four hours are usually very deceiving. All patients in this class are conscious and rational. They usually refuse transportation by stretcher. Shivering is invariably present. There is complaint of more or less severe pain. Pulse and temperature are usually within normal limits, but the blood pressure is below normal. The white cell count shows an increase. During the first twelve to twenty-four hours the red cell count, hemoglobin and blood plasma protein determinations are within normal limits, but shortly after this time the red cell count and hemoglobin begin to show marked increases while the plasma proteins decrease. The signs and symptoms of secondary shock rapidly become manifest. The patient is usually irrational, the pulse is rapid and weak, the respirations are labored, and death usually intervenes within six to ten hours.

The apparent lack of signs and symptoms of circulatory disorder within the first twelve to twenty-four hours may be explained by the compensatory mechanisms coming into play in an effort to maintain blood volume because of the pronounced loss of blood plasma due to capillary permeability. Thus it is imperative that all therapeutic efforts be directed toward offsetting or minimizing the peripheral circulatory failure that is inevitable if appropriate measures are not instituted long before signs and symptoms appear.

### Treatment

All therapeutic effort should be directed toward combating, as soon as possible, inevitable shock, the intensity of which will depend upon the severity and urgency of the emergency.

*Rest.*—Immediately following an accident, the victim should be transported from the scene by means of a stretcher—the first aid in minimizing shock. A hypodermic injection of morphine sulfate (grain  $\frac{1}{4}$ ) is administered as soon as pos-

sible to minimize pain, and this is repeated in doses of  $\frac{1}{8}$  to  $\frac{1}{6}$  grain every three to four hours in order to keep the patient comfortable.

*Body Heat.*—The patient, who is generally shivering should as soon as possible be wrapped snugly in blankets to keep up body warmth. If available, hot water bags or a heating pad are applied while he is awaiting transportation to a hospital. Butter, lard, and other fats and oils add to the possibility of future infection and are not used on the burned areas. The hospital bed should be covered by sterile sheets and the patient surrounded by an electric light cradle for supplying heat. The opening of large blisters is to be recommended but any extensive débridement may add to shock and therefore if indicated it should be put off for about forty-eight hours.

*Vital Supportive Therapy.*—The replacement of blood plasma proteins is imperative before the signs and symptoms of circulatory failure appear. The intravenous administration of *blood plasma* is to be started as soon as possible. Initial doses of as much as 1000 cc. have been given, with an additional 500 cc. every six hours until 4000 to 6000 cc. have been administered. The initial dose and the total quantity are dependent upon the severity of the emergency. The commercially dried human blood plasma may be conveniently dissolved and readily administered in any locality, its only disadvantage being the cost. It is particularly ideal for administration to a patient who has to be fortified before being transported a long distance during which much valuable time would otherwise be lost. It is to be remembered that for the purpose of combating capillary permeability so as to keep the plasma proteins within the vascular beds and thereby maintain blood volume, subcutaneous injections of *adrenal cortex extract* (2 cc. every three hours) are very highly recommended.

In those cases in which a generous number of plasma infusions and an occasional blood transfusion were administered, the use of intravenous solutions of 5 per cent *glucose* and *normal saline* is usually restricted to amounts varying from 750 to 1500 cc. per day; however, large amounts up to tolerance are administered by other routes.

A prophylactic dose of 1500 units of *tetanus antitoxin* administered subcutaneously should be a routine measure in the early treatment of burns.

*Topical Applications.*—In severe burns local applications of astringents, antiseptics and analgesics are made to the burned areas for the purpose of (1) alleviating pain, (2) preventing absorption of burned tissue toxins, (3) minimizing secondary infection, and (4) preparing for future skin regeneration or replacement.

As soon as possible following the first plasma infusion, a freshly made 5 per cent aqueous *tannic acid* is sprayed upon the burned areas every one to two hours until a firm eschar has developed. Great care is taken not to aggravate shock by disturbing the patient unduly. Some practitioners prefer additional applications of 10 per cent *silver nitrate* to the edges of tannic acid eschars. In other medical circles, applications of 1 per cent gentian violet on cotton swabs are preferred to the tannic acid spray. There are many commercial preparations on the market claiming various advantages for their application to burns; however, their efficacy, especially in severe burns, is unproved.

Applications of *warm saline* or *boric acid packs* to soothe painful areas are important. To cleanse infected wounds, irrigations with diluted *Dakin's solution* are employed. For loosening and removing adherent sloughs, a warm, continuous flow bath with force spray that burrows underneath sloughs and washes out purulent pockets is valuable. Very little bleeding occurs from this procedure.

Measures to prevent future contractures should be started early.

#### EXPOSURE TO ABNORMALLY HIGH OR LOW ATMOSPHERIC TEMPERATURES

In the human, emergencies frequently occur from exposures to environmental temperatures, the intensities of which are markedly above or below that of the body temperature. While body heat is normally regulated within extremely narrow limits, it is maintained in these limits even in the presence of wide fluctuations of environmental temperatures by

means of the intricate mechanisms of (1) chemical regulation or heat production, and (2) physical regulation, or heat loss. However, in the presence of organic disease, such as arteriosclerosis and other disorders which interfere with heat dissipation, serious emergencies may arise from body heat retention resulting in hyperpyrexia. Profuse and prolonged perspiration, with low salt intake, may cause collapse and subnormal temperature due to the resulting extremely low blood chlorides. On the other hand, exposures of a sparsely clad person to environmental temperatures much below that of the body temperature may give rise to emergencies because of increased heat dissipation and insufficient heat production resulting in a hypothermia.

### Heat Stroke

A prolonged exposure to high atmospheric temperatures ( $100^{\circ}$  to  $138^{\circ}$  F.), excessive humidity (70 to 100) together with a lack of air currents, may give rise to serious and urgent emergencies, the severity of which will depend upon how high the consequent body temperature rises ( $105^{\circ}$  to  $112^{\circ}$  F.). The higher the body temperature, the more serious is the prognosis, and therefore, the greater the urgency for temperature reduction at the very earliest opportunity.

The emergency is essentially one of *intoxication*, and the resulting fever is due to the absorption of imperfectly split proteins caused by abnormally low water intake.

**SIGNS AND SYMPTOMS OF AN EMERGENCY.**—The first sign of impending danger is the *cessation of perspiration*. The skin is intensely hot and usually dry. As the rectal temperature passes  $106^{\circ}$  F., delirium, stupor, or coma develops. While the muscles are usually relaxed, fibrillary twitchings and even convulsions may occur. As the temperature continues to rise, coma deepens. Patients in whom the rectal temperature reaches  $109^{\circ}$  F. and cannot be reduced within several hours, seldom survive. An early fall in rectal temperature and a regaining of consciousness is considered very favorable.

**TREATMENT.**—The patient should be disrobed to the underwear, placed in the recumbent position, and moved to some cool, shady place. *Ice bags* are applied to the head and both

axillae. The use of hand fans to stir up air currents is advisable. At the home or hospital, immediate efforts must be made to lower the body temperature. At the St. Louis City Hospital, the most effective method has been the *ice tub immersion*, accompanied by brisk rubbing. However, in less experienced hands, an *ice cold wet sheet* enveloping the nude patient is recommended. An air blast from an electric fan should be directed across this sheet which must be dampened frequently.

It is very important to take the rectal temperature every two minutes because the temperature should not be allowed to descend below 102° F. On account of a possible "temperature bounce," all efforts at reduction should stop when 103.5° F. is reached. It sometimes happens that due to a treacherous temperature bounce, the temperature continues to descend until 97° F. is reached. When this occurs, efforts must be made to raise the temperature back to 102° F. by means of hot blankets and water bottles. During the temperature reduction, a circulatory stimulant, such as *caffeine sodiobenzoate* (grains 2 to 6), injected intramuscularly every three hours is recommended. If the pulse is excessively fast, an intravenous injection of *digalen* (1 ampule) repeated every three to six hours may be advisable.

The unconscious patient should have water and salt as soon as possible. The intravenous or subcutaneous administration of *normal saline* in a 5 per cent dextrose solution in quantities of 500 to 1000 cc. repeated every four to six hours is recommended. The conscious patient should be given increasing quantities of *cold water* by mouth along with *sodium chloride tablets* (15½ grains) every three hours.

### Heat Exhaustion

Prolonged exposures and vigorous muscular activity in environments of high atmospheric temperature and humidity may give rise to a train of signs and symptoms leading from *heat cramps* to an emergency called *heat collapse*.

Heat collapse is a mild form of heat stroke. The body temperature, instead of being high as in heat stroke, is either normal or subnormal (it may fall as low as 95° F.). The sur-

face of the skin is pale, cold, clammy and moist, the pulse is weak and rapid, and respirations are shallow and rapid. Pupils are dilated. In severe attacks, there may be delirium or unconsciousness, and the delirium may lead to coma. Death seldom occurs unless the patient has been seriously ill before the heat exposure.

**TREATMENT.**—For the *conscious patient* provide lightweight and loose-fitting clothes and place in a cool place with good air circulation. Give cool drinks of fruit juices, and sodium chloride (grains 15) every three hours in either plain or enteric-coated tablets.

In the case of the *unconscious patient*, if the rectal temperature is subnormal, wrapping in light blankets with a hot water bottle at the feet is recommended. The administration of normal saline with or without 5 per cent glucose solution by either one or both the intravenous and subcutaneous routes should be started as soon as possible. Caffeine sodio-benzoate (grain 1) subcutaneously every three hours is considered of benefit as a circulatory stimulant. When the patient regains consciousness further treatment is symptomatic.

### Freezing

Exposures of a part or the entire surface of the body to environments of near or subzero temperatures, especially when improperly protected by clothing or other insulation, may give rise to serious emergencies. The resulting emergency is primarily one of nutritional impairment of the tissues, the extent of which will depend upon the environmental temperature, the duration of the contact and the surface area involved. The types of lesions produced by these contacts are classified into (1) local freezing, and (2) general freezing.

**LOCAL FREEZING.**—The type and extent of the lesions resulting from contacts with low environmental temperatures are divided into three classes:

**First Degree.**—The areas most frequently involved are the tip of the nose, lobes of the ears, toes and fingers. The parts affected at first give rise to a transient stinging pain and soon become white and insensitive. An individual seldom realizes the condition until warned by some passer-by who may rub

axillae. The use of hand fans to stir up air currents is advisable. At the home or hospital, immediate efforts must be made to lower the body temperature. At the St. Louis City Hospital, the most effective method has been the *ice tub immersion*, accompanied by brisk rubbing. However, in less experienced hands, an *ice cold wet sheet* enveloping the nude patient is recommended. An air blast from an electric fan should be directed across this sheet which must be dampened frequently.

It is very important to take the rectal temperature every two minutes because the temperature should not be allowed to descend below  $102^{\circ}$  F. On account of a possible "temperature bounce," all efforts at reduction should stop when  $103.5^{\circ}$  F. is reached. It sometimes happens that due to a treacherous temperature bounce, the temperature continues to descend until  $97^{\circ}$  F. is reached. When this occurs, efforts must be made to raise the temperature back to  $102^{\circ}$  F. by means of hot blankets and water bottles. During the temperature reduction, a circulatory stimulant, such as *caffeine sodiobenzoate* (grains 2 to 6), injected intramuscularly every three hours is recommended. If the pulse is excessively fast, an intravenous injection of *digalen* (1 ampule) repeated every three to six hours may be advisable.

The unconscious patient should have water and salt as soon as possible. The intravenous or subcutaneous administration of *normal saline* in a 5 per cent dextrose solution in quantities of 500 to 1000 cc. repeated every four to six hours is recommended. The conscious patient should be given increasing quantities of *cold water* by mouth along with *sodium chloride tablets* ( $15\frac{1}{2}$  grains) every three hours.

### Heat Exhaustion

Prolonged exposures and vigorous muscular activity in environments of high atmospheric temperature and humidity may give rise to a train of signs and symptoms leading from *heat cramps* to an emergency called *heat collapse*.

Heat collapse is a mild form of heat stroke. The body temperature, instead of being high as in heat stroke, is either normal or subnormal (it may fall as low as  $95^{\circ}$  F.). The sur-

sis demand immediate and high amputations in order to save life.

**GENERAL FREEZING.**—Excessive exposure of the insufficiently clad individual to low environmental temperatures will develop an emergency, the urgency of which will depend upon how low the body temperature falls. At first, the person has muscular weakness, stiffness of the limbs, and shows signs of fatigue. As the body temperature goes lower, drowsiness and apathy set in, together with irresistible desire to sleep. If the person is discovered before he freezes to death in this sleep, there is a good chance of recovery, which is brought about by gradual warming in a cool room together with massage of the entire body area with cool cloths. Artificial respiration may be necessary. Warm enemas of plain water or black coffee will help in raising the body temperature. Immersion in a bath the temperature of which is gradually raised from 60° to 85° F. over a period of two hours is recommended by some authorities. Stimulants such as strychnine sulfate (grain  $\frac{1}{60}$ ) or caffeine sodiobenzoate (grains 2) by hypodermic every three hours are invariably indicated. It must be remembered that even after a return of consciousness and normal body temperature there is a danger of sudden death.

#### INJURIES FROM CONTACTS WITH ELECTRICITY

The degree of emergency arising from contacts with electricity will depend on a variety of factors such as (1) type and intensity of electrical current, (2) extent of the contact on the body, (3) duration of the contact, and (4) body resistance and grounding.

There are three kinds of electrical currents which offer possibilities of producing accidents, namely (1) low tension currents, (2) high tension currents and (3) lightning.

##### Contacts with Low Tension Currents

Low tension currents consist of two types, the AC or alternating current, and the DC or direct current. Both of these currents have a pressure of 110 to 115 volts and an intensity of 10 amperes, but, all other factors being equal, the one most

the parts briskly with snow in order to terminate the exposure with slow thawing. Under no circumstances should the patient be taken immediately to a warm atmosphere—slow thawing should be continued either in the open or the coolest part of the house. Hands and feet are best thawed out by immersing in cold water (50° F.) accompanied by gentle massaging, with encouraged movement of the parts. In the thawing-out process there is stinging pain and a reactionary hyperemia which is usually followed in a day or two by edema of the parts involved.

*Second Degree.*—Involvement of larger areas and continued exposure results in the parts becoming gray-white, hard, insensitive and motionless. When the parts are gradually thawed as above mentioned, the areas are very painful and develop a red to very dark red color over which blisters usually form in several days. Raw or encrusted areas should be treated aseptically. A prophylactic dose (1500 units) of *tetanus antitoxin* should be injected when these lesions are present. If gentle massage together with cold applications do not bring about the desired tissue color changes, alternate occlusion by a blood pressure cuff pumped to 80 mm. for ten to twenty seconds and then released—compression and release repeated three to five times per minute—may be practiced. Passive vascular exercise (pressure and suction machine) has been recommended if available especially when the patient must be brought to warm quarters at the earliest opportunity.

*Third Degree.*—When the measures advocated for the slow thawing of frozen extremities fail, the condition is considered to be of third degree. After attempts at thawing, the part while still remaining a yellowish-white to a bluish-gray color is cold and insensitive, but usually looks much better than the emergency warrants. If no blisters form in the next several days, gangrene, either of the dry or wet type, is present. The wet type is considered the more dangerous on account of toxin absorption. Further treatment becomes a major surgical problem. Owing to progressive necrosis of the anoxic tissues, some delay may be encountered in the demarcation of a true line for amputation. However, cases which show sep-

from the lungs and the gastric contents; (2) its ease and efficiency of administration.

The victim is rolled over on the abdomen. One arm is extended and the forearm is flexed at the elbow, the head is placed on this hand and the face is turned to the opposite side. The operator, facing the head in a kneeling position, then straddles the patient and places each hand with the fingers extending towards the sides over the floating ribs. Then in cycles of ten to fourteen per minute, the force is exerted so that the pressure on the diaphragm forces air out of the lungs and then a sudden release allows the atmospheric pressure to rush air into the lungs. The administration of pure oxygen or carbon dioxide and oxygen mixtures, by means of face mask, funnel, or plain rubber tubing, should accompany this procedure as soon as it is available. A pulmotor or any form of "iron lung" should carry on as soon as either one is available. If there was any possible hope of revival, the chances of resuscitation will depend upon how soon artificial respiration was started.

### High Tension Currents

Contact with electrical currents of ultra-high voltage (2200 and higher) and amperage (50 amperes and higher), of the alternating type, may produce grave emergencies and are apt to cause immediate death. With the alternating current there is a greater tendency for the subject to "freeze" to the contact, so that along with a greater initial shock there is a more severe burn (charring) at the site of contact. In those accidents where by coincidence the person is thrown away from the contact, accompanying lesions such as contusions, lacerations, dislocations and fractures are possible.

If death is not immediate, the person is always unconscious. The heart rate may be rapid or imperceptible but the respirations are at a standstill and death may be the result of oxygen-want (anoxia) of the tissues if the respirations are not restored within eight minutes. Freeing of the patient from the electrical circuit and immediate artificial respiration are the first principles of emergency treatment. When accidents of this nature happen on high tension lines, linemen are trained

apt to cause fatal or serious injury is the alternating current because contact with it produces tetanic contractions in muscles resulting in severe initial shock and, more or less, in an extensive burn at the site of entry of the current. The production of tetanic contractions in the muscles will result either (1) in "freezing" the subject to the point of contact because of a flexor spasm of muscles, or (2) in his being thrown away from the point of contact, with possible resulting trauma.

**SYMPTOMS.**—The signs and symptoms of electrical shock will depend upon the severity of the emergency. If the contact and ground, or conductor, are good, the person will be rendered unconscious by the shock and respiration and heart beat apparently cease, which may mean an instantaneous death.

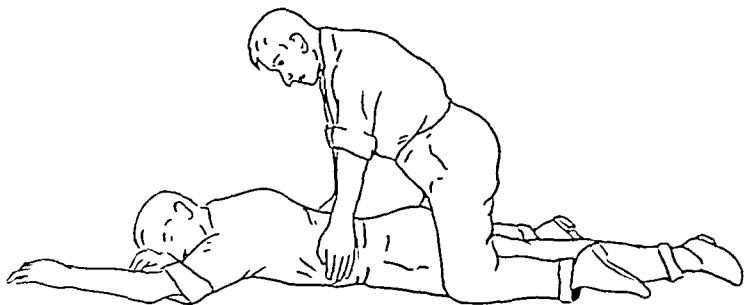


Fig. 72.—Position of patient and operator in the Schaefer method.

However, a spark of life may be present which can be revived by immediate and prolonged *artificial respiration* by means of the Schaefer method, and pulmotor or respirator, along with inhalations of carbon dioxide and oxygen. The possibility of revival, therefore, will depend not only on how small and short the contact was but also upon how soon and for how long artificial respiration was administered.

**Schaefer Method of Artificial Respiration.**—While there are many and various methods with and without apparatus used for resuscitation, the one most commonly administered and recommended is the Schaefer method. The particular advantages of this method are: (1) the position of the head causes the tongue to drop away from the back of the pharynx, thus promoting a better passage for air and for expelling fluids

gen through the tissue capillaries will cause death in a much shorter period of time.

#### Decreased Atmospheric Pressures

Altitudes of 20,000 feet are about the limits to which an unacclimatized individual can ascend without an artificial supply of oxygen. At 25,000 feet unconsciousness usually occurs. Rapid ascents by airplane or balloon to these upper levels may result in a sudden loss of consciousness due to a reduction of oxygen supply to the brain. Flyers of fast pursuit ships, preliminary to an ascent, inhale pure oxygen in order to get a high concentration in the lungs, and by means of a special face mask and a special supply of oxygen endeavor to maintain in their lungs an oxygen concentration of approximately 50 per cent. These rapid ascents without oxygen offer another problem by producing nitrogen bubbles around nerve sheaths, as in the "bends," which are caused by the rapid change to a lowered atmospheric pressure. This condition, however, is very effectively prevented by the preliminary and the continued inhalations of oxygen during a flight.

In altitudes of approximately 18,000 to 20,000 feet where consciousness is not lost, a serious or fatal emergency may develop from the foolhardy and ill-judged acts of an aviator who has developed a mental and sensory dullness, muscular weakness, headache, vomiting, dyspnea and cyanosis due to the diminished supply of oxygen to the brain, which is the first tissue to feel the want of oxygen. Dropping quickly out of these altitudes brings the individual back to full senses. However, an extra oxygen supply administered by face mask is the best preventive.

#### Decreased Oxygen Concentrations

Emergencies will develop in those individuals shut in airtight compartments, such as bank vaults, refrigerators and submarines, if the oxygen supply is gradually consumed without replenishment. When the blood oxygen concentration drops to 13 per cent, cyanosis develops, but when a concentration of 8 per cent is reached the individual is in grave oxygen want and may die shortly from suffocation.

to administer artificial respiration by the "pole top resuscitation method" so as to save the precious minutes necessary to lower the injured to the ground for the Schaefer method. However, on the ground, the Schaefer method, pulmotor, and the respirator are the methods of choice. Efforts at resuscitation must be continued for hours even though the outcome looks very gloomy.

### Injuries by Lightning

The majority of these accidents result in immediate death. However, lightning sometimes plays queer pranks. After striking a tree in a rainstorm, it may just graze a person standing beneath it, partially disrobing him by tearing off a trouser leg and removing one or both shoes. The person is rendered unconscious, which may be of variable duration. Respiration may stop temporarily or be left irregular and labored. The skin is usually cold and clammy as in collapse. Heart rate is rapid and thin. Mild to medium burns, ecchymoses and muscle tremors are usually present.

**TREATMENT.**—Artificial respiration, the maintenance of normal body temperature by heat and blankets, and other forms of supportive therapy are imperative in the emergency treatment.

As a measure of the last resort, an injection of  $\frac{1}{2}$  cc. of *adrenalin* into the ventricle of the heart by puncture through the chest wall and heart muscle is recommended. If no adrenalin is available and all other measures have failed, the chest wall may be punctured with a large needle, and through the opening an attempt is made to initiate heart action by scratching or irritating the heart muscle with the needle, using an up-and-down stroke.

### ASPHYXIA

Emergencies arising from accidents in which there is an interference with oxygen utilization within the body are rather frequent. The urgency of these emergencies is most serious in view of the apparent lack of adequate oxygen storage—death will result in eight minutes if access to an outside source of oxygen is completely cut off. Any general interference, as by a histotoxic substance, to the diffusion of oxy-

Inhalations of minute quantities of the gas will produce dizziness and headache while increased quantities cause marked muscular weakness and unconsciousness accompanied by rapid, stertorous breathing, a dusky hue of the skin and blue or pale lips. In coma there may be marked muscular rigidity, particularly of the jaws. The patient dies of anemic anoxemia.

For treatment the patient should be removed to an atmosphere of fresh, warm air. Artificial respiration is started immediately, together with inhalations of 95 per cent oxygen and 5 per cent carbon dioxide, and a stimulant such as coramine or metrazol is given by hypodermic injection as soon as possible. The intravenous injection of medicinal methylene blue (50 cc. to a 1 per cent solution in 500 cc. of 5 per cent glucose) may be of some help. Blood transfusions are sometimes indicated. If consciousness is not restored in several hours the prognosis is exceptionally grave.

#### *Interference with Normal Oxygen Diffusion in the Capillaries*

The ingestion or inhalation of histotoxic substances, especially certain cyanide compounds like hydrocyanic acid and the oil of bitter almonds which interfere with the normal diffusion of oxygen through the tissue capillaries, will produce grave emergencies. The gravity of the emergency becomes apparent when it is realized that tissue cells will not live one moment without free oxygen; consequently, complete interference will result in sudden death.

*Hydrocyanic Acid Poisoning.*—Free hydrocyanic acid is the principal constituent of the gas used in fumigating buildings. Inhalation of this gas through its seepage into nearby living quarters or by a too early entrance into fumigated quarters will produce an emergency, the gravity of which will depend upon the amount of gas inhaled. Large doses produce sudden death. With small doses, the signs and symptoms are dizziness, headache and extreme shortness of breath. The pulse is weak and slow. There is a peach odor to the breath and the mouth is usually covered with foam. In coma the respirations become slower and cease before cessation of the heart beat. The patient dies of histotoxic anoxemia.

Pure oxygen supplied through an emergency opening will save an infant until a rescue is effected.

### Obstructions to Air Passages

Most prominent of the obstructions to the air passages are *asphyxia* of the lungs due to the inhalation of dense air smoke and suffocation from being covered by clothing. A pure emergency will result if the obstruction is complete because death will intervene within eight minutes. A complete or near-complete obstruction by an object of the lungs must be relieved by immediately with a finger thumb or other instrument within eight or ten minutes if the individual is asphyxiated. A near-complete obstruction by foreign substance minutes of 30 per cent better and 20 per cent oxygen may be administered by inhalation. Various forms of "iron lung" with and without oxygen and carbon dioxide inhalation arrangements have an important place in these emergencies especially when they are produced by a paralysis of the muscles of respiration.

### Interference with the Normal Oxygen-carrying Power of the Blood Stream

Emergencies arising from an interference with the normal oxygen-carrying power of the blood stream may be caused by a wide variety of agents, the most important of which are carbon monoxide.

*Carbon Monoxide Poisoning.*—Emergencies arising from the inhibition of carbon monoxide are the results of oxygen want in the blood stream. Anemia caused by a decrease of the blood hemoglobin in the production of carbon monoxide poisoning causes poisoning the formation of carbon monoxide hemoglobin. The extent of the emergency depends on the quantities of carbon monoxide inhaled and the duration of the exposure. Quantities of 1 part of carbon monoxide to 1000 parts of air will produce headache in an hour and unconsciousness in two hours while concentrations of 11 parts to 1000 will produce unconsciousness and death in two hours. Greater quantities will cause death in much shorter periods of time.

Inhalations of minute quantities of the gas will produce dizziness and headache while increased quantities cause marked muscular weakness and unconsciousness accompanied by rapid, stertorous breathing, a dusky hue of the skin and blue or pale lips. In coma there may be marked muscular rigidity, particularly of the jaws. The patient dies of anemic anoxemia.

For treatment the patient should be removed to an atmosphere of fresh, warm air. Artificial respiration is started immediately, together with inhalations of 95 per cent oxygen and 5 per cent carbon dioxide, and a stimulant such as coramine or metrazol is given by hypodermic injection as soon as possible. The intravenous injection of medicinal methylene blue (50 cc. to a 1 per cent solution in 500 cc. of 5 per cent glucose) may be of some help. Blood transfusions are sometimes indicated. If consciousness is not restored in several hours the prognosis is exceptionally grave.

#### Interference with Normal Oxygen Diffusion in the Capillaries

The ingestion or inhalation of histotoxic substances, especially certain cyanide compounds like hydrocyanic acid and the oil of bitter almonds which interfere with the normal diffusion of oxygen through the tissue capillaries, will produce grave emergencies. The gravity of the emergency becomes apparent when it is realized that tissue cells will not live one moment without free oxygen; consequently, complete interference will result in sudden death.

*Hydrocyanic Acid Poisoning.*—Free hydrocyanic acid is the principal constituent of the gas used in fumigating buildings. Inhalation of this gas through its seepage into nearby living quarters or by a too early entrance into fumigated quarters will produce an emergency, the gravity of which will depend upon the amount of gas inhaled. Large doses produce sudden death. With small doses, the signs and symptoms are dizziness, headache and extreme shortness of breath. The pulse is weak and slow. There is a peach odor to the breath and the mouth is usually covered with foam. In coma the respirations become slower and cease before cessation of the heart beat. The patient dies of histotoxic anoxemia.

Emergency treatment: In unconsciousness action is very urgent and is best accomplished by two persons with medical experience. Artificial respiration must be started immediately together with inhalations of amyl nitrate for ten to twenty seconds every two to three minutes. Intravenous injection of sodium nitrite (0.3 gm. in 10 cc. of water) should be followed immediately by 50 cc. of a 50 per cent sodium thiosulfate solution through the same needle. Oxygen inhalations are oftentimes necessary. The application of external heat is indicated if subnormal body temperature is present.

Ingestion of cyanide will require additional measures, such as gastric lavage with an antidote of 3 per cent hydrogen peroxide or a 0.2 per cent potassium permanganate solution. Survival will depend upon the amount ingested and the length of time intervening from the accident until emergency measures were started.

#### BIBLIOGRAPHY

- Blalock, Alfred: Principles of Surgical Care, Shock and Other Problems. C. V. Mosby Co., St. Louis, 1940.
- Fisher, Ellis Hart: Arch. Phys. Therapy, 10: 611 (Oct.) 1940.
- Trusler, H. M., Egbert, H. L. and Williams, H. S.: Burn Shock: Blood Chemical Studies and Report of an Extensive Burn Treated by Repeated Transfusions of Blood and Blood Plasma. J.A.M.A., 113: 2207, 1939.
- Wilson, W. C., Jeffrey, J. S., Rowley, G. D. and Gray, N. A.: Acute Toxemia of Burns. Extract of Suprarenal Cortex in Treatment. Lancet, 1: 1400, 1936.
- Harkins, H. N. and Harmon, P. H.: Thermal Injuries: Effects of Freezing. J. Clin. Invest., 16: 213, 1937.
- McConnell, W. J.: Diseases due to Physical Agents. Cecil Textbook of Medicine, W. B. Saunders Co., Philadelphia, 1940.

## THE DIAGNOSIS OF TUBERCULOSIS IN GENERAL PRACTICE\*

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To anyone conversant with the tuberculosis problem it might appear rather trite to claim that therapeutic measures in the disease usually are more efficacious and meet with better end-results when the diagnosis has been made in the earliest stage. Failure to make a correct early diagnosis in this widespread disease is very frequently fraught with serious consequences. Not only does it materially affect the ultimate well-being of the one so afflicted but frequently is of untold harm to members of the immediate family and to all individuals who come into intimate daily contact with the patient.

Tuberculosis is still one of our major health problems and is the leading cause of death in our most useful and productive age groups. It is responsible for fully one seventh of all deaths and kills about one third of all who die between the ages of fifteen and forty-five. The recognition of the early case of tuberculosis is the best weapon we have at our command to combat this disease. Early diagnosis does not depend on any one method nor is it necessary for the physician to be a specialist. To make a diagnosis of tuberculosis it is essential that the physician be *thorough* and *methodical* in his examination of the patient. This examination in order to be thorough must be based on the following well known and time-tested procedures: (1) careful history; (2) complete physical examination; (3) x-ray examination; and (4) laboratory procedures, consisting of sputum examination, determination of the sedimentation rate, tuberculin test and blood examination.

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## HISTORY

The information derived from a carefully taken history is often sufficient, without other aid, to suggest the proper diagnosis. The most important elements in such a history are the *symptoms* that led the patient to seek medical advice, and the careful elicitation and correct interpretation of these early subjective symptoms by the attending physician should be considered of prime importance. The signs that are brought out by physical examination or the data that are obtained by the aid of a laboratory come into the diagnostic picture long after the early subjective symptoms have made their presence felt. In the average case the physician will not ordinarily have a sputum examination made or a roentgenogram taken unless these procedures are indicated to verify a suspicion aroused by their presence. Experience shows that unless the physician sees these early symptoms in their true light, much time is lost and the patient fails to obtain a correct diagnosis until the disease is well advanced.

From a practical viewpoint the best classification of the symptoms of pulmonary tuberculosis is one that is based on etiology. Such a classification was suggested a number of years ago by Pottenger<sup>1</sup> (Table 1). The table embraces practically all the symptoms and signs that one will encounter not only in the early stage of tuberculosis but also in the moderately or far advanced stages. With the exception of a positive sputum, no single symptom is definitely pathognomonic of the earliest stage. However, the presence of two, three or more of the symptoms mentioned would be highly suggestive of such a condition.

From a clinical viewpoint it is obvious that the importance of any single symptom would be to a great extent dependent upon the incidence of its relative frequency in a large series of cases. To determine the relative frequency with which these symptoms make their earliest appearance a survey of 133 patients undergoing treatment at Mt. St. Rose Sanatorium during a period of one year, was made<sup>2</sup> (Table 2). A study of this table reveals that *cough* and *weakness* occurred most frequently, and that *gastro-intestinal symptoms*, *lack of endurance* and *loss of weight* were also common. Frequently it

TABLE 1

ETIOLOGIC GROUPING OF COMMON SYMPTOMS OF PULMONARY TUBERCULOSIS

Group I Symptoms due to Toxemia	Group II Symptoms due to Reflex Cause	Group III Symptoms due to the Tuberculous Process per se
Malaise	Hoarseness	Frequent and pro-
Lack of endurance	Tickling in larynx	tracted colds (tu-
Loss of strength	Cough	berculous bronchi-
Nervous instability	Digestive disturbances	tis)
Loss of appetite	which may result in	Spitting of blood
Digestive disturbances	loss of weight	Pleurisy (tuberculosis
(hypomotility and hy-	Circulatory disturb-	of pleura)
poscretion)	ances	Sputum with or with-
Metabolic disturbances	Chest and shoulder	out bacilli
resulting in loss of	pains	
weight	Flushing of face	
Increased pulse rate	Spasm of muscles of	
Night sweats	shoulder girdle	
Elevation of temperature	Diminished motion of	
Anemia	affected side. Lag-	
Leukocytic changes	ging	
	If chronic, degenera-	
	tion of apical soft	
	tissues	

TABLE 2

MOST FREQUENT EARLY SYMPTOMS OF PULMONARY TUBERCULOSIS

Symptoms	Number of Cases	Per cent
Cough .....	53	39.0
Weakness .....	50	37.6
Gastro-intestinal disturbances .....	21	15.7
Lack of endurance .....	21	15.7
Loss of weight .....	19	14.2
Pleurodynia or pleurisy .....	12	9.0
Hemorrhage .....	11	0.8
Fever .....	9	0.6
Nervous instability .....	5	0.3
Hemoptysis .....	5	0.3
Night sweats .....	3	0.2
Hoarseness .....	3	0.2
Dyspnea .....	1	0.75

was noted that patients were treated symptomatically for long periods of time ranging from a few weeks to six or nine months without any apparent effort being made to ascertain the cause of their symptoms.

Williams and Hill<sup>3</sup> made an analysis of pulmonary tuberculosis in 1499 white patients over fifteen years of age. They discovered that only 17 per cent were classified in the minimal or early stages at the time of admission to twelve different sanatoria. Their investigations also revealed that the five most frequent symptoms causing patients to seek medical advice were, in the order of their appearance and relative importance, as follows:

1. Cough accompanied or not by expectoration or an acute respiratory cold, 1309, or 87 per cent.
2. Lack of endurance or being too easily tired, 1245, or 83 per cent.
3. Loss of weight, 1115, or 74 per cent.
4. Loss of appetite, 805, or 53 per cent.
5. Pain in the chest, pleuritic or otherwise, 693, or 48 per cent.

*Hemoptysis* and *pleurisy* with effusion while presumptive evidence of tuberculosis were present in a comparatively small number of cases. *Hemorrhage* in their statistics ranked eighteenth in importance.

These studies serve to emphasize the importance of a thorough knowledge of symptoms in the early recognition of this disease by the physician. The majority of patients seek advice early but unfortunately their condition is not always thoroughly studied or correctly diagnosed. Too, some practitioners hesitate to inform the patient that he is tuberculous. This is a serious mistake. We have never seen a patient die from the shock of being told that he has tuberculosis, but we have observed numbers of patients who have died because they were not informed in time.

#### Elements of a Good History in Suspected Tuberculosis

*Chief Complaints.*—Frequently the sole symptom complained of by the patient is hemoptysis or hemorrhage. In other instances he may mention several symptoms, such as anorexia, loss of weight and cough. The importance of symptoms has already been discussed.

*Onset of Present Illness.*—Having obtained all possible information concerning the patient's present complaints, the physician proceeds to elicit the full details of the illness by painstaking and careful questioning. He attempts first to ascertain the time of onset of the patient's illness. Very frequently patients do not recognize insignificant symptoms until their attention is called to them by the doctor. As a result of such questioning, one can in most instances discover that the disease has been developing over a much longer period of time than the patient realizes. After making a complete study of the duration and gradual development of the present illness the physician frequently has a clear-cut mental picture of what he will find on physical examination.

*Personal or Past History.*—Now the physician endeavors to develop the background of the patient's past life. This may have been entirely devoid of illness, or there may have been recurrent attacks of colds, pleurisy and other respiratory infections. The patient may have been more or less sickly for many years prior to his present trouble. This information may or may not confirm the facts already brought out in the history.

*Family History.*—The importance of family history in its relation to tuberculosis has been greatly overemphasized. Experienced clinicians see many instances of active pulmonary tuberculosis in persons whose family is free from the disease and many others in which all other members of a family remain free from manifest tuberculosis for many years, after one member of that family has succumbed to the disease. These facts, however, should not cause the examiner to neglect this otherwise important phase of the examination. Not infrequently when we discover that a member of a patient's family has suffered from so-called "chronic bronchitis" or asthma, we are able to prove that the individual has actually had a tuberculosis which was not diagnosed. The finding of unrecognized tuberculosis in a member of a patient's family has frequently been demonstrated in tuberculosis surveys. When children are found to be positive reactors to tuberculin tests it has been frequently demonstrated that an open case of unrecognized tuberculosis exists in the family.

Finally, the family history gives us important information regarding the social and economic background of the patient.

*Habits.*—Habits play an important role in the well-being of an individual and unless they are carefully gone into by the physician he may miss an important clue that will aid in the making of a correct diagnosis.

During the past two decades we have observed the widespread and one might say almost universal addiction to the *cigarette habit* by the public. Not infrequently a patient comes to the doctor's office complaining of loss of appetite, loss of weight, nervousness and a chronic cough. His friends, or even his family doctor, may have suggested the possibility of early tuberculosis. No evidence of active tuberculosis is found, but upon closely questioning the patient in regard to his cigarette habit, we are told that he is smoking and inhaling from twenty to sixty per day. When he gives up or greatly curtails the habit the symptoms disappear.

The practitioner should also obtain clear-cut data concerning the amount and kind of *alcohol* that patient imbibes. This is important as its immoderate use may have a definite bearing on treatment and prognosis.

*Occupational History.*—The vast majority of patients who develop tuberculosis are engaged in seeking a livelihood, and it is necessary to ascertain the character of work they do. Pulmonary conditions like silicosis and anthracosis may simulate tuberculosis and have frequently been mistaken for it.

## PHYSICAL EXAMINATION

### Physical Signs

A few important physical signs are usually present in active cases and can be elicited by simple tests.

*Fever.*—Given a patient with history of persistent cough, weakness and loss of appetite and weight, we should always obtain a chart of his temperature and pulse taken at two-hour intervals during the day from 8 A.M. to 8 P.M. for several days. In such a patient an afternoon rise of temperature to 99° F. or above is strongly indicative of active tuberculosis, provided careful search fails to reveal other cause for the fever.

*Pulse.*—With the temperature chart a record of the pulse

should be made, since the pulse rate is an even more sensitive indicator of tuberculous toxemia than is the temperature. However, since the pulse rate is influenced by emotional disturbances, mental reactions and endocrine disorders to a marked degree, one should be very cautious in using an elevated pulse rate as a sign of tuberculous toxemia in the absence of fever. The relationship between pulse rate and fever is, however, highly significant. A rather fast pulse of 100 or more per minute with a low grade afternoon fever is of utmost diagnostic value.

*Loss of Weight.*—Loss of weight, while not peculiar to the toxemia of tuberculosis, is such a constant and characteristic feature of the disease that the weight should always be determined.

*Anemia.*—The color and general appearance of the patient with early activity of a tuberculous lesion are apt to be very good, while marked anemia and cachexia may be interpreted as indicators of the disease which is relatively advanced both as to time and gravity of the toxemia.

#### Examination of the Chest

This important phase of the examination in suspected tuberculosis is oftentimes carried out cursorily by the busy practitioner. To be satisfactory, the best results are achieved by having the patient comfortably seated before the examiner with his chest completely exposed. Then inspection, palpation, percussion and auscultation should be methodically and carefully performed, step by step in the order mentioned. The amount of information obtained will depend entirely upon the degree of skill and the experience of the examiner. Expertness in this field cannot be fully attained merely by reading textbooks upon the subject; constant study and application of the involved principles are requisites. Although statistics compiled in the Trudeau Sanatorium by Brown and Sampson, based on 280 cases of minimal tuberculosis, reveal that positive physical findings (rales) are present in only 27 per cent of cases, whereas positive x-ray evidence is present in 99 per cent, these findings should not discourage us from giving careful attention to the steps which follow.

*Inspection.*—By inspection we study the *general appearance* of the subject and the *contour* of the thorax. A search is made for evidence of an underlying pathologic process, evidence consisting of inequality in the size of the two sides of the chest, undue prominence of a clavicle, or retraction of an intercostal space. The respiratory excursion is studied and its rate is counted. If there is a lag in the excursion on one side as compared with the other, we may consider the *inequality of expansion* as due to one or both of two factors: (1) active disease with muscle spasm and consequent fixation, (2) an old lesion with fibrosis of lung and pleura and atrophy of the muscles of the chest wall.

The location of the *apex beat*, if visible, is noted. It may be displaced to the right or the left of its normal position. If displaced to the *left* it may indicate retraction due to fibrosis or atelectasis of the left lung parenchyma with compensatory emphysema in the contralateral lung, or a pleural effusion or pneumothorax of the right lung. If displaced to the *right* the pathology may be reversed. Frequently by causing the subject to swallow one can observe a deviation of the trachea towards the diseased side of chest. In early cases, inspection may reveal very little if any change and the examiner then proceeds to the next step.

*Palpation.*—This method of diagnosis, which when properly conducted may yield valuable information, is often neglected. The best technic in my experience is to place the right hand, with fingers outstretched and separated, firmly in the interspaces of the anterior surface of one side of chest, and simultaneously to place the left hand on the posterior surface while having the subject pronounce in a low-pitched voice "ninety-nine" or "one, two, three." This will best bring out an increase, decrease, absence of or normal fremitus, depending on whether we are dealing with a diseased lung. At times because of heavy musculature or obesity this procedure may prove unsatisfactory. No definite information is obtained in early cases, but in moderately or far advanced conditions there are usually definite changes. Palpation of the muscles of the shoulder girdle and the intercostal muscles will frequently reveal the presence of *muscle spasm* on one or both sides. This

pathologic change as demonstrated by Pottenger is the result of a reflex disturbance, and is always an indication of a disease process in the underlying lung parenchyma. It bears the same relation to lung disease as a rigid abdominal rectus muscle does to acute appendicitis.

*Percussion.*—In extremely early tuberculosis this procedure may not give much information. The results of percussion depend on the type of anatomic changes occurring in the lung and naturally will show great variations in individual cases. As the tuberculous process in the majority of patients first makes its appearance in the apices or upper portions of the lung, special attention should be given to these areas when percussing. *Unilateral apical dullness* is one of the most frequent signs in pulmonary tuberculosis. It is generally most distinct in the highest intercostal spaces anteriorly, but sometimes in the early stages it is found only in the infraclavicular fossa or more often at the back of the supraclavicular fossa. As the infiltration progresses the dullness becomes more extensive.

To obtain the best results from percussion, it is necessary for the examiner to use a very light percussion stroke and to cover systematically the anterior, posterior and lateral surfaces of the chest wall. I employ the following technic, which is at variance with most textbook teachings. The first step is to lightly percuss both clavicles and compare the sounds. If there is any disease in either apex the dullness of the one side will be definitely contrasted with the resonant note of the healthy contralateral side. The next step is the percussion of the right lung anteriorly from its apex downward, which is accomplished with the index finger of the left hand firmly in the intercostal spaces. By a comparison of the percussion notes obtained in each succeeding interspace it is possible to outline any area of dullness present. This procedure should be carried out anteriorly, posteriorly and laterally, first on one side and then on the other. The outlines of the area of dullness are most easily ascertained by contrasting the hyperresonant note usually found in the lower portion of the lung field with the impaired resonance usually found in the upper portion of the lungs in tuberculosis.

*Auscultation.*—While this procedure yields no signs that are definitely pathognomonic of tuberculosis, yet it may be said that the extent of the tuberculous disease in the lung can be defined more accurately by means of auscultation than by any other method of physical diagnosis. The tuberculous process produces anatomic changes in the lung parenchyma which give rise to adventitious breath sounds whose type and character depend on the extent of the process. The most common auscultatory signs are crepitations or *rales*, first described by Laënnec.

*Rales* are any abnormal sounds heard within the lung during respiration, and may be classified as *dry* or *moist*, according to the absence or presence of fluid in the air passages. The *dry rales* are characterized as sibilant, sonorous and rhonchal. They are produced by narrowing of the lumen of the air passages in acute or chronic inflammatory processes and are of common occurrence in such conditions as acute bronchitis, asthma, bronchial tuberculosis, bronchiectasis and malignancy of the lungs.

Moist rales are divided into fine, medium, large or coarse and mucous rales. Fine rales, also known as crepitant rales, probably originate in the air cell in the presence of exudate or increased moisture, and may also be caused by the separation of an atelectatic air cell when its walls are forceably separated by deep breathing. Medium rales or subcrepitant rales occur in the smaller bronchioles under similar conditions, their presence denoting a widespread area of involvement. The coarse, mucous or large rales occur in the larger bronchioles and denote a still further extension of the disease process within the lung parenchyma.

The earliest physical sign of clinically active tuberculosis is the presence of *fine moist rales* usually heard in a small area in the upper third of either lung field. They definitely denote an active disease process and are the most important factor in the detection of early tuberculosis. They can best be elicited by instructing the subject to cough at the end of expiration, the cough to be immediately followed by deep inspiration. If the lesion is extensive these rales can be heard on ordinary deep breathing. *Bronchial* and *amphoric breathing* denote ex-

tensive infiltration and cavitation, which of course are present only in the more advanced conditions.

### X-RAY EXAMINATION

Today the x-ray is the mainstay in the diagnosis of early pulmonary tuberculosis. It will often reveal the presence of an early lesion long before physical signs make their appearance. Every patient suspected of having tuberculosis should have an x-ray film taken and undergo a fluoroscopic examination. The fluoroscope, while not showing details of peribronchial thickening and other changes as clearly as does the x-ray plate, nevertheless reveals gross changes, extent of diaphragmatic movement, chest expansion, and the extent to which the apices are aerated on deep inspiration. By judicious use of both x-ray methods the extent, distribution and character of all lesions, early as well as advanced, can definitely be determined and studied. Moreover, the information thus gained is vital to the clinician in his conduct of a therapeutic regimen. No phthisiologist today would attempt to carry out treatment whether by absolute bed rest or collapse methods without resorting to the x-ray for guidance during the course of treatment.

### LABORATORY PROCEDURES

*Sputum.*—The demonstration of acid-fast organisms in the sputum is still the only direct and specific evidence of clinically active tuberculosis. A specimen of sputum if obtainable should be examined in every suspected case. A single negative specimen should not be considered as final evidence of the absence of active tuberculosis, if the preponderance of findings points the other way. Repeated examinations may be necessary and for a final diagnosis guinea-pig inoculations may have to be resorted to. With laboratory facilities at our command today throughout the country, in rural as well as in larger communities, the simple though essential sputum examination should never be neglected in suspected cases.

*Sedimentation Rate.*—This relatively new procedure has become universally recognized as a test to determine the presence of an active infection. It is not specific for tuberculosis but it

does reflect the course and progress of the infection when routinely employed during treatment.

*Schilling Differential White Cell Count.*—This is another method which, while not specific for tuberculosis, will give valuable information as to its course and frequently is of value from a diagnostic standpoint.

Leukocytosis is a sign of activity. If the increase in the number of white cells consists chiefly of young immature elements, the sign is unfavorable, particularly if it includes the appearance of myelocytes and is associated with an absolute decrease of the lymphocytes. On the other hand, if the increase in the number of white cells consists chiefly of a lymphocytosis, the reaction is a more favorable one. A marked lymphocytosis is a sign of healing.

*The Erythrocyte Count.*—This important procedure should be carried out routinely in all cases whether or not a diagnosis has been established. It is of great value in revealing the presence of a blood dyscrasia such as secondary anemia, pernicious anemia, or agranulocytosis.

*Other Blood Studies.*—A *hemoglobin* determination should be made and the *color index* should always be estimated. Occasionally it may be necessary to make a *blood culture* to determine the presence or absence of a blood stream infection. The information thus gained may often be helpful in differentiating a subacute bacterial endocarditis from an early tuberculosis.

*Tuberculin Test.*—Since the introduction of tuberculin as a diagnostic aid by von Pirquet, the technic has been variously modified and today the Mantoux method of intracutaneous injection is almost universally employed. Another variation is known as the *skin patch test*. These tests find their greatest usefulness in case finding. A negative reaction denotes the absence of tuberculous infection, while a positive one indicates its presence, though not necessarily in a clinically active form. Thus a negative reaction is definitely more valuable than a positive one. This test is not indicated as a routine measure, but when the diagnosis is questionable it may be distinctly helpful. The principal use of this test is in large tuberculosis surveys, where the following up of positive re-

actors may lead to the uncovering of unrecognized cases in the reactors' families.

### CONCLUSION

In the foregoing paragraphs the writer has endeavored to outline briefly the essential steps in the diagnosis of pulmonary tuberculosis in the early stages. As previously stated, the general practitioner is not required to be an expert in this disease but he must know the elementary and fundamental principles of diagnosis. Above all he must be tuberculosis-minded and consider the possibility of this disease in every instance in which the symptoms and signs are vague and indefinite. The careful application of this idea will often reward the doctor with the recognition of an early case which otherwise would have gone undiagnosed.

Lawrason Brown<sup>4</sup> once stated that he would like to see a placard in every physician's office prominently displayed, reading: "Remember syphilis and tuberculosis. These are the two great simulators of other diseases. The parasites of each work under cover, in darkness, and when they become easy to detect, the ravages of the disease are frequently so extensive that hope of recovery is gone and only a temporary arrest is possible. The detection of such a foe demands all the diagnostic acumen that one can command and, indeed, only when it is kept constantly in mind can it be detected early."

### BIBLIOGRAPHY

1. Pottenger, F. M.: The Cause of the Various Clinical Manifestations in Pulmonary Tuberculosis. *Ann. Int. Med.*, 2: 20 (July) 1928.
2. Henske, A. C. and Ehlers, C. W.: Early Symptoms of Pulmonary Tuberculosis. *Missouri State Med.*, April, 1931, pp. 164-167.
3. Williams, L. R. and Hill, A. M.: *J.A.M.A.*, 93: 579 (Aug. 24) 1929.
4. Brown, Lawrason: The Diagnosis of Pulmonary Tuberculosis for the General Practitioner. *J.A.M.A.*, 90: 1032 (March 31) 1928.



## TREATMENT OF TUBERCULOSIS IN GENERAL PRACTICE\*

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THE successful treatment of pulmonary tuberculosis taxes to the utmost the skill, intelligence and patience of the attending physician. In many diseases the physician may adopt, as the result of his experience, a routine practice, modified chiefly by the degree of severity of the symptoms. However, this does not hold true in phthisis, for there is no malady which assumes so many protean forms and is attended by such diversified complications. The incipency of this disease is frequently hidden in a long train of vague symptoms, sometimes extending over many years; while, again, it strikes an apparently normal individual with the suddenness and the violence of an acute infection. In either case it is apt to follow a varied course of alternate exacerbations and arrests over a long period. There is no disease which more sorely tries the fortitude and constancy of the patient as well as that of the physician.

Prognosis in this condition is still very uncertain at times, both as to the final outcome and duration of the disease. It is, therefore, plainly evident that at the very inception of treatment the relations of the attending physician and patient should be based upon *mutual understanding*. There is nothing to be gained and often much that is important to the well-being of the patient may be lost, by making light of the infection, no matter at how early a stage it is recognized.

Under the most favorable circumstances the course of treatment will, by the very nature of the disease, be carried out over a long period of time. One cannot assume that any pa-

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tient is cured, no matter how benign or early his disease was when treatment was begun, until two to three years have elapsed since all symptoms of the infection have disappeared. This fact should be kept in mind by the physician and from the very beginning of treatment be clearly impressed upon the patient. It is the first requisite to intelligent medical management of the disease.

The facts upon which advice and treatment will depend in any given case are in the order of their importance: (1) the stage and duration of the disease; (2) the financial condition of the patient; (3) his social condition, previous habits, and temperament; (4) his age; and finally, his (5) occupation.

With these facts determined the physician is then enabled to advise to the best advantage whether the patient is to be (1) sent to a sanatorium, (2) treated at home, or (3) treated in a dispensary.

Whether the patient is to be separated from his family and whether he is to be permitted to attend in whole or in part to his business are related matters to be settled. Each patient is an individual problem and his personal requirements will to some extent modify the course of treatment. In fact there is no condition in which *individualization* plays a more important role in achieving a successful outcome than in tuberculosis. This holds true in the sanatorium as well as in the home or in the clinic.

The *fundamental factors* in the treatment of pulmonary tuberculosis are (1) rest, (2) diet, (3) suitable hygienic surroundings, fresh air, etc., and (4) collapse therapy. All these measures are to be carried out under strict, intelligent medical supervision.

#### SANATORIUM TREATMENT

Thirty years ago the practice of sending tuberculous patients to hospitals or sanatoria was in its infancy, and institutions for their care were few and far between. Today there is hardly a county or a state that does not have a number of sanatoria to provide for the treatment of tuberculous patients. These institutions are usually supported by their communities and the only qualification for admission is that the patient be a bona fide resident of the locality. The goal of two beds for

every tuberculosis death, the minimum requirement set up by the National Tuberculosis Association years ago, is today a reality.

It is conceded by all authorities that, whenever possible, a tuberculous subject should be given the advantage of at least a preliminary course of treatment and instruction in a sanatorium. In no other way will he so readily learn the value and rationale of strict adherence to a therapeutic regimen. In the past, institutions limited their admission to patients with early or moderately advanced lesions, that is, to subjects who had a reasonable chance for recovery. Today, however, if there are sufficient beds in a community or state, patients in all stages of the disease are admitted regardless of their condition and regardless of their financial circumstances. The theory is that a patient thus isolated from his family and immediate associates is one less source of contagion and infection.

At present the wide use of collapse therapy and thoracic surgery has made absolutely essential the hospitalization of most patients at some time during the course of their treatment.

#### HOME TREATMENT

While it is conceded by everyone that hospitalization in most instances is ideal, yet for various reasons, financial or otherwise, it is practically impossible to have all patients taken care of in sanatoria. Sometimes, too, home treatment is actually to be preferred to hospital care, provided the surroundings are satisfactory and suitable nursing care can be obtained. The writer has in the past handled many cases in the home successfully and can see no valid reason why such a procedure cannot be recommended when circumstances warrant it. The same rules and regulations can be carried out as well in certain homes as in an institution.

When artificial pneumothorax is indicated, however, the patient should receive his initial treatments in a hospital. This is essential because of the necessity of x-ray examinations to determine the success or failure of pneumothorax treatment. After a pneumothorax is satisfactorily established in the hospital, then there is no objection to the patient receiving further treatment in the home.

## DISPENSARY OR CLINIC TREATMENT

The role of the dispensary or clinic in the treatment of this disease concerns itself chiefly with the ambulatory patient who has been discharged from the sanatorium with quiescent or apparently arrested disease. The clinic through its social service department should also function as a health center in the community for the dissemination of knowledge and information pertaining to tuberculosis, and as a center for periodic health examinations of the indigent, for follow-up work in suspected cases and for examination of members of a patient's family and other individuals who give a recent history of exposure. The clinic is a necessary and important adjunct to the municipal sanatorium or semiprivate hospital, providing as it does the after-care of indigent patients who are discharged from these institutions and continue to require observation, guidance and pneumothorax refills. In this manner the dispensary or clinic contributes to the successful recovery and rehabilitation of many patients who have had the misfortune to become victims of this disease.

## CLIMATE

There was a time in the past when the profession believed that a change of climate was one of the most important factors in the successful treatment of tuberculosis. This belief has gradually been dispelled, owing to our growing consciousness of the benefits of absolute bed rest and collapse therapy. Many patients who in the past were advised to go west by their family physicians in the hope that such a change of environment would bring about a cure, unfortunately failed to get well, because climate alone is not sufficient to bring about such a result. Bed rest and hygienic-dietetic measures under the supervision of a physician are necessary adjuncts. One might say that the value of climate as a therapeutic measure can be roughly estimated at about 5 per cent. Observation has demonstrated that a patient can recover, regardless of climate, provided he obtains good medical supervision. Furthermore, the sending of patients to a distant part of the country, away from their family and associates, often has a demoralizing effect on the individual and incurs for him a heavy monetary

expense. In the past many unfortunate patients have spent their time, money and energy in seeking from place to place what is really unobtainable—a climate which will *cure* tuberculosis.

#### HYGIENIC-DIETETIC TREATMENT

To Brehmer is credited the inauguration of the hygienic-dietetic treatment at Goerbersdorf, Germany, in 1859. It consists essentially in placing the patient in the best possible environment for increasing resistance and in counteracting the effects of toxins and waste by maintaining nutrition at its highest efficiency. It is the rational method and, as has been said, treats the patient as well as the disease. This method was elaborated in Brehmer's sanatorium and has provided the fundamental principles of all treatment of tuberculosis whether practiced in the institution or in the home. It consists of (1) an almost constant life in the open air; (2) rest and exercise apportioned to the individual and modified from time to time according to clinical indications; and (3) a properly balanced diet suited to individual requirements.

Brehmer's original method has since been somewhat modified and improved upon. The first detail to be changed was the method of *rest* and *exercise*. Detweiler, who was a pupil and former patient of Brehmer, observed that patients frequently had relapses and he believed that these followed as a result of exercise taken before the patient had had a sufficient period of rest in bed. He proved to his own satisfaction that the underlying factor in successful treatment by the method is *absolute bed rest* over a sufficient period of time, until all clinical evidences of the disease have disappeared; then, and only then, is the patient to be placed on graduated exercise.

The next change brought about was in our conception of the so-called "*fresh-air treatment*." There was a period not so many years ago when it was thought that living out in the open air was one of the essential prerequisites for getting well. As time went on it was observed that it was not so much the fresh air as good ventilation that was important, and that a well ventilated room served the same purpose.

Finally, the last change came about in regard to the *diet*. Years ago patients were literally placed upon forced feeding,

with the result that in many instances there were gastrointestinal upsets. Too much stress was laid upon the importance of eggs and milk to the neglect of the other essential constituents of a well balanced diet as conceived today. Thanks to our increasing knowledge, patients are now placed on a well balanced diet containing 2500 to 3500 calories per diem depending on their weight, stage of disease and age. In addition, proper attention is given to the requirements for vitamins and mineral salts.

### COLLAPSE THERAPY

Observation and experience also taught the profession that, despite the careful carrying out of the foregoing measures, many patients did not fully respond to treatment. It was frequently noted that patients with cavitation were the ones who usually gave the most trouble. Notwithstanding gain in weight and disappearance of clinical symptoms many of these patients continued to have positive sputum and were subject to recurrent acute exacerbations with spread of the disease to other parts of the remaining healthy lung. They became known in the profession as the so-called "good chronics," and the outlook for their complete recovery or cure was, to say the least, discouraging.

These observations gradually led to the development of *collapse therapy* as a necessary and vitally important adjunct to bed rest in the treatment of pulmonary tuberculosis. It has been only during the last twenty-five years that this procedure has become fully recognized as a great step forward in the treatment of this disease. This belated recognition was probably due to the early failure on the part of the general profession to diagnose tuberculosis in the early stages, and to the lack of the universal use of the x-ray. Today these factors do not obtain. The fact that nowadays no physician should consider a diagnosis of pulmonary tuberculosis complete without the use of the roentgen ray films as an integral part of a complete history, gives the diagnostician a better understanding of the extent of the pathologic process, and enables him to visualize more clearly the type of treatment which may in a given case be productive of the most satisfactory results.

## MANAGEMENT OF CLINICALLY ACTIVE PULMONARY TUBERCULOSIS

When the diagnosis has been established, the lesion should be classified under one of the following groups: (1) minimal, (2) moderately advanced, (3) far advanced, or (4) terminating.

## Minimal Lesions

In minimal cases the lesion is confined to a small part of one or both lungs, the total volume of involvement, regardless of distribution, not exceeding the equivalent of the volume of lung tissue which lies above the second chondrosternal junction and the spine of the fourth or the body of the fifth thoracic vertebra on one side.

Regardless of the stage of disease the patient should immediately be placed upon *absolute bed rest* for at least *three months*. The importance of rest can best be illustrated by the fact that the respiratory rates of normal persons on absolute bed rest have been found to be between 8 and 12 less per minute than when these persons engaged in slight or active exercise. This reduction of respiratory work in the daily continuous functioning of the lungs can be estimated to amount roughly to about 480 respirations per hour and 5760 per twenty-four hours. This enormous reduction in work of the lungs on absolute bed rest is the physiological explanation why tuberculous lesions tend to heal rapidly in the vast majority of instances, when nature is given an opportunity to assert itself. Fever, which is an expression of toxemia and a constant symptom of activity, gradually subsides, and resolution of the disease process within the lung parenchyma takes place. The appetite improves, weight is gained and cough if present gradually disappears. This in turn favorably influences the body defence mechanism, thus enabling the patient to overcome his infection. Experience has proved that the minimal period required to bring about such a result is three months. In many instances in which the pathologic process is further advanced the rest period may have to be extended to six to twelve months.

During the bed-rest regimen the patient should be exam-

ined carefully at frequent intervals to determine whether the physical signs in the lung are clearing. If physical examination does not reveal this tendency, x-ray studies at intervals for definite evidence of regression or progress are necessary. The early minimal lesion will usually respond satisfactorily to bed rest alone, provided a suitable, well balanced diet, carefully figured out to meet the needs in a given case, is employed. Cough, if not productive, can be controlled with a sedative such as dilaudid, grain  $\frac{1}{64}$ , or codeine sulfate, grain

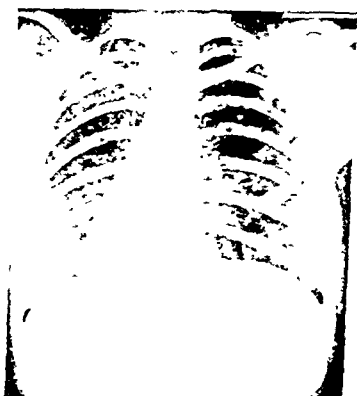


Fig. 73.



Fig. 74.

Fig. 73.—Miss E. S. (6/17/41). Moderately advanced unilateral pulmonary tuberculosis involving the right upper lobe. No evidence of cavitation. Positive sputum.

Fig. 74.—Same patient four months later (10/22/41). Film reveals the results of absolute bed rest. Sputum negative. Sedimentation rate normal. Patient up and on exercise. Apparently quiescent case.

$\frac{1}{6}$  per dose. If after three to six months of bed rest the physical signs of activity and all subjective symptoms have cleared and x-ray and laboratory findings, such as sedimentation rate and sputum are confirmatory, the patient can be placed on a course of graduated exercises. These should be increased until the patient is able to be up and about at least six hours during the day without any appearance of reactivity. He then is instructed to walk gradually increasing distances daily over a period of two months until he is walking about one mile

daily morning and afternoon. If there is no reactivation of the disease process the case can then be considered quiescent or apparently arrested, depending upon the degree and extent of the pathologic process at the time of beginning of treatment. The patient has now arrived at the point where he may be permitted to resume on part time his former occupation, provided it is a sedentary one and not heavy manual labor. In the case of manual laborers it is advisable to rehabilitate the patient and induce him to change to a lighter form of work.

During the next six months the patient while working should be kept under strict medical supervision so that any evidence of reactivation, should it appear, can be promptly detected and checked by whatever measures the attending physician deems advisable.

The results to be expected from a regimen of bed rest in properly selected cases are shown in Figs. 73 and 74.

#### Moderately Advanced Lesions

The moderately advanced lesion involves one or both lungs, and is more widely distributed than the minimal lesion, varying in extent, according to the severity of the disease, from the equivalent of one third the volume of one lung to the equivalent of the volume of an entire lung with little or no evidence of cavity formation. No serious tuberculous complications are present.

Here likewise, *absolute bed rest* is an essential therapeutic measure and should be carried out in a similar manner but perhaps over a longer period of time than in the treatment of the first stage. It is also advisable to supplement bed rest with *artificial pneumothorax*. This measure should as a rule be initiated immediately, because, if one defers too long, the development of pleural adhesions will either prevent the establishment of a satisfactory collapse or interfere with the complete collapse of cavities. It must be understood that the primary purpose of a pneumothorax is to collapse cavities and unless this purpose is attained there is no valid reason for continuing a pneumothorax. Experience has taught that unless cavities are obliterated, it is practically impossible to attain a negative sputum. As long as the sputum remains positive, one

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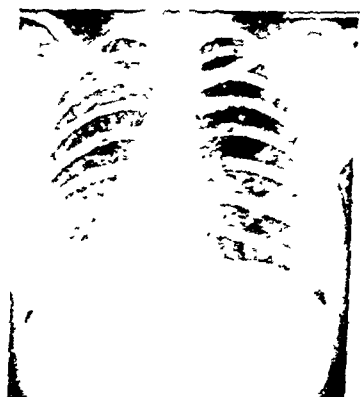


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is dealing with an open case that may at any time flare up and spread to adjacent areas of healthy lung parenchyma.

Having once established a successful pneumothorax, the progress of the patient will usually be satisfactory. The same rules as outlined in the treatment of minimal cases can then be applied. Frequently a patient can be permitted to return to his former occupation, anywhere from six to twelve months after the treatment has been started.

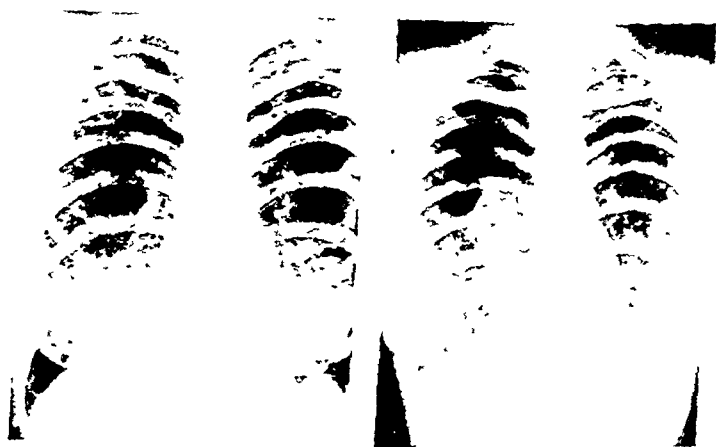


Fig. 75.

Fig. 76.

Fig. 75.—Miss A. M. (3/25/41). Moderately advanced pulmonary tuberculosis with large cavity in right upper lobe, second and third interspace.

Fig. 76.—Same patient four months later (7/22/41). Film shows a successful right selective pneumothorax, with closure of cavity in right apex. Apparently quiescent case with negative sputum and normal sedimentation rate.

*How long an artificial pneumothorax should be continued* is still a mooted question and can be decided only by a careful evaluation of the character and extent of the pathologic process in a given case at the time treatment is begun. Generally speaking, one may state that artificial pneumothorax should not be terminated in less than one year. In the average case refills may be required for one to three or four years before the patient is finally discharged as recovered. (See Figs. 75 to 78.)



Fig. 77.



Fig. 78.

Fig. 77.—Miss V. A. (3/29/40). Far advanced bilateral pulmonary tuberculosis with large cavities in both upper lobes.

Fig. 78.—Same patient about eighteen months later (9/4/41). Film reveals a successful bilateral pneumothorax. Apparently quiescent case with negative sputum and normal sedimentation rate.

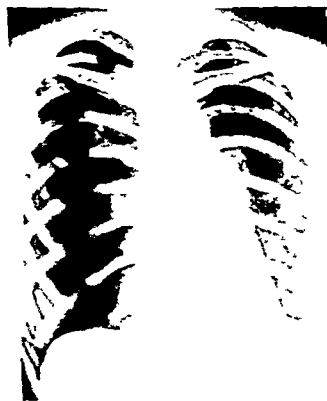


Fig. 79.



Fig. 80.

Fig. 79.—Miss M. B. (3/31/41). Left pneumothorax unsatisfactory, because of adhesions preventing complete collapse of cavity in left upper lobe. Positive sputum and still evidence of slight activity.

Fig. 80.—Same patient approximately five months later (8/16/41), shortly after intrapleural pneumonolysis was performed. A satisfactory collapse of right upper lobe with complete obliteration of cavity has been obtained. Sputum negative. Sedimentation rate normal. Apparently quiescent case.

Occasionally, adhesions prevent complete collapse of the cavity (Fig. 79). To overcome this, Jacobaeus devised a procedure known as *intrapleural pneumonolysis*. By this method with the aid of a specially devised instrument, the thoracoscope, the operator is enabled to enter the pleural space and with an electrocautery sever adhesions that are preventing the complete collapse of cavities. The procedure can be carried out successfully in a large percentage of cases, thereby converting an incomplete and unsatisfactory pneumothorax into a successful one (Fig. 80).

### Far Advanced Lesions

The far advanced lesion is more extensive than the moderately advanced, or it shows definite evidence of marked cavity formation, or there are serious tuberculous complications.

Here successful treatment becomes more difficult because of the duration and extent of the pathologic process. The morale of the patient may be undermined, resulting from repeated failures towards recovery in the past. Nevertheless, *bed rest* plays an important role and during a period of observation the physician must decide as to what one of the various procedures at his disposal is to be employed to combat the infection. Artificial pneumothorax either unilateral or bilateral (Figs. 77 and 78), phrenic nerve crushing, artificial pneumoperitoneum or thoracoplasty may be resorted to, either in combination or successively. Occasionally the involvement is confined to one lung but more frequently in this stage it is bilateral with one lung more involved than the other. If feasible an effort should be directed towards controlling the disease in the lesser involved lung. This can often be accomplished with a unilateral pneumothorax, a phrenic nerve crushing, or in some instances by artificial pneumoperitoneum. Some times it may be necessary to carry out all three of these procedures in an individual case.

*Phrenic Nerve Crushing.*—This is an operation whereby one leaf of the diaphragm is paralyzed by severing or crushing the phrenic nerve at its most accessible point, just above the clavicle in the cervical region of the side on which the leaf of the diaphragm is to be elevated. This procedure immobil-

izes the affected leaf, causing it to elevate from one to three interspaces, thereby reducing the apicobasal diameter of the chest (Figs. 81, 82).

Phrenic nerve crushing has its most striking effect in the treatment of partially stabilized lesions in which there are one or more isolated thin-walled cavities not surrounded by too much infiltration. It produces relaxation of the surrounding structure which may promote healing and may also be a great aid in controlling hemorrhage.

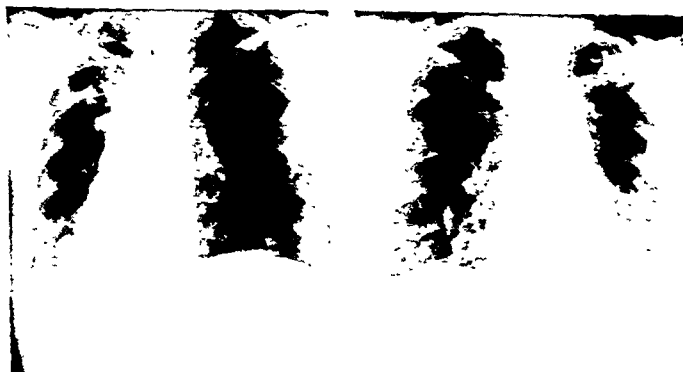


Fig. 81.

Fig. 82.

Fig. 81.—Miss D. F. (2/13/40). Moderately advanced pulmonary tuberculosis with large cavity in left apex. Pneumothorax unsuccessful.

Fig. 82.—Same patient eight months later (10/8/40), following a left phrenic crushing operation which resulted in marked elevation of left leaf of diaphragm, causing obliteration of cavity in left apex. Sputum negative. Sedimentation rate normal.

*Artificial Pneumoperitoneum.*—By this method air is introduced into the peritoneal cavity. This causes an increase in the intra-abdominal pressure thereby elevating both leaves of the diaphragm and limiting to some degree their excursion during respiration (Figs. 83, 84). This treatment is a measure of last resort, to be used when all other procedures are contraindicated. It frequently has a beneficial effect in otherwise hopeless cases and often brings about improvement in the less affected lung, so that the other procedures may at times later be carried out.

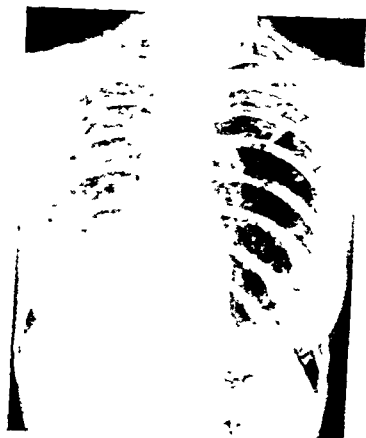


Fig. 83.



Fig. 84.

Fig. 83.—Miss M. S. (1/17/40). Far advanced bilateral pulmonary tuberculosis with large cavity in right upper lobe. Pneumothorax unsuccessful.

Fig. 84.—Same patient about ten months later (11/2/40). Artificial pneumoperitoneum has resulted in a marked rise in right leaf of diaphragm to level of eighth rib and on left side to tenth rib. There has been a marked clearing up of the lesions with almost complete disappearance of cavity in right apex.



Fig. 85.



Fig. 86.

Fig. 85.—Mr. T. P. (11/14/39). Moderately advanced pulmonary tuberculosis involving right upper lobe with cavitation. Pneumothorax unsuccessful.

Fig. 86.—Same patient eighteen months later (5/21/41). Two-stage right thoracoplasty. This is an arrested case with obliteration of cavitation, negative sputum and normal sedimentation rate.

*Thoracoplasty.*—Thoracoplasty is a purely surgical procedure introduced by Sauerbruch and Brauer of Germany a number of years ago. It is carried out by an extrapleural resection of the ribs over the affected part of the lung, so as to release the attachments and tensions which may be preventing the closure of cavities and to exert positive compression to accelerate this process. There are various modifications of this procedure. It has its most striking effect in the treatment of



Fig. 87.

Fig. 87.—Mr. J. H. (4/22/40). A tuberculous pneumonia involves the right upper lobe.



Fig. 88.

Fig. 88.—Same patient ten months later (2/22/41), revealing the effects of a successful right pneumothorax instituted immediately upon admission to hospital. At present case is apparently arrested, with sputum negative and a normal sedimentation rate.

partially stabilized lesions in which there is an isolated thin-walled cavity not surrounded by too much infiltration. It also produces relaxation of the surrounding structures which may promote healing. The operative risk in the hands of trained surgeons is less than 5 per cent. Satisfactory results in well selected cases vary between 70 and 90 per cent.

This operation offers to many patients the hope for a recovery and return to a useful and comfortable life, when other measures have been found wanting (see Figs. 85, 86).

### Symptomatic Treatment

Cough, fever and anorexia are symptoms that usually improve or disappear, when once the patient has been placed upon bed rest. Occasionally, as previously stated, a sedative may be required to control an unproductive cough. One of the most important and dramatic symptoms is hemoptysis or hemorrhage which frequently is the source of much concern to both the patient and the physician. When severe it can be controlled by inducing artificial pneumothorax on the affected side or, if this is impossible, an artificial pneumoperitoneum in the writer's experience has proved highly satisfactory.

It must be remembered that the severity of symptoms is often in direct relation to the extent of pathologic process, and therefore the symptoms are more of a therapeutic problem in the far advanced and terminating cases. Here at best, treatment can be only palliative and the physician can only to some degree relieve the discomfort and suffering of the patient by judicious handling of the case.

### PROGNOSIS

It is very difficult to formulate any general statement regarding prognosis in this disease. The prognosis should always be guarded, lest a too optimistic opinion may plague the physician in the event of an unfavorable outcome. As a general rule one may state that the earlier the diagnosis is made, the more favorable is the chance for recovery provided proper and adequate therapeutic measures are immediately instituted. This rule will apply in the vast majority of cases with the exception of tuberculous pneumonia, florid phthisis, miliary tuberculosis, tuberculous meningitis and unforeseen accidents, such as pulmonary hemorrhage.

In arriving at a prognosis one must also take into consideration the age, habits, social environment, economic status and the general physical constitution of the patient. These factors may influence for better or worse the outcome in a given case.

### CONCLUSIONS

While it is not necessary that a physician be an expert to diagnose tuberculosis, he does require long experience and

mature judgment to treat pulmonary tuberculosis satisfactorily in all its phases. With the rapid advances that have been made within the past two decades in tuberculosis therapy, it has to a great extent become a highly specialized field in therapeutics.

However if tuberculosis like smallpox is ever to be eradicated, it will not be due to our efforts in treatment. The highest aim of the medical profession should be the *prevention* of disease and its spread. At present the profession has at its command the knowledge and scientific methods with which the prevention and complete control of tuberculosis are possible, but unfortunately, for various reasons, we are not fully applying our knowledge to this end. Like the farmer, we lock the stable door after the horse is stolen. A recent report given out by a United States Government bureau stated that bovine tuberculosis had been completely eradicated in this country during the past ten years. If this be so, why cannot the same results be achieved in the eradication of human tuberculosis in this country? If such a humanitarian plan as a mass survey were adopted and carried out, tuberculosis like smallpox would become within a generation a disease of the past.



## MANAGEMENT OF THE COMPLICATIONS OF TUBERCULOSIS\*

H. I. SPECTOR, M.D., F.A.C.P., F.A.C.C.P.†

THE management of the complications of pulmonary tuberculosis in its broadest concept should be considered under five main headings, as indicated in the discussion which follows.

### I. COMPLICATIONS RESULTING FROM THE CON- TINUED ADVANCEMENT OF THE TUBERCULOUS PROCESS

#### 1. Pleurisy with Effusion

For the sake of proper treatment, pleurisy with effusion must be classified according to the character of the effusion, which may be serous or bloody, sero-purulent, or purulent, and also according to whether it is the result of a tuberculous or a mixed infection.

Pleurisy with effusion may manifest itself as an *initial symptom* in tuberculosis with little or no accompanying pulmonary disease, or it may occur later in the course of the disease incidental to pneumothorax treatment. The management of an effusion will depend on the symptoms of the patient, the massiveness, the bacteriologic characteristics, and on whether or not underlying pulmonary disease exists, whether or not a bronchial fistula is present, and on the condition of the opposite lung. Specimens of all accessible effusions occurring in a patient for the first time should be obtained and studied grossly, microscopically, chemically, culturally and histologically, and through the medium of guinea pig inoculation, if necessary, for the purpose of determining whether or not

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there exists a pure tuberculous or a mixed infection empyema. All massive effusions, regardless of the complicating bacteriologic organisms, should be removed in sufficient quantity to relieve symptoms, thus permitting visualization of the compressed lung. When definite underlying pulmonary disease exists, it may be advisable to replace the fluid with air and thus establish a therapeutic pneumothorax depending on the condition of the opposite lung. When no evident pulmonary disease is present, the patient should be advised to rest in bed for a period of from three to six months depending on the symptoms in the individual case, regardless of whether tubercle bacilli are found in the pleural fluid.

When the underlying lung lesion is controlled, a tuberculous serous or purulent empyema developing *during the course of pneumothorax therapy* is not so serious a complication as was formerly considered, since spontaneous healing with absorption of the fluid not infrequently takes place with modified bed rest alone. Such an empyema associated with a *bronchopleural fistula*, however, presents a serious complication.

*Mixed infection empyemas* usually result either from infection from without through repeated tapplings or from within through the medium of spontaneous or traumatic pneumothorax. This type of complication presents a real problem in management and has an unfavorable influence on the prognosis. Injection into the pleural cavity of a .05 to 1 per cent watery solution of either gentian violet or crystal violet has been helpful in some cases. These solutions have a marked effect on the gram-positive organisms. For the gram-negative ones Dakin's solution or azochloramid, a chlorine compound somewhat weaker than Dakin's solution, has been reported to have beneficial results. Rib resection followed by thoracoplasty occasionally becomes a necessity, especially when a bronchopleural fistula exists. Generally, uncomplicated effusions should be left alone since many heal spontaneously.

## 2. Spontaneous Pneumothorax

Spontaneous pneumothorax may manifest itself as an initial symptom or may appear during the course of the disease,

especially during the pneumothorax treatment. The management of such a complication will depend on the suddenness of onset and on the rapidity and completeness of the compression. If the opening in the visceral pleura is large, and the lung is too rapidly compressed, immediate and frequent *removal of air* from the pleural cavity is necessary in order to relieve symptoms. *Oxygen therapy* may at times be indicated, and *morphine* in doses of  $\frac{1}{8}$  to  $\frac{1}{4}$  grain or *codeine*  $\frac{1}{4}$  to 1 grain, may be necessary to control the pain and the cough. Patients who have a large opening in the visceral layer of the pleura present the problem of marked dyspnea and symptoms of shock generally. In such patients the dyspnea is controlled to some extent by inserting a *catheter* into the pleural space, the tube being so placed that it opens slightly below the level of the patient into a weak antiseptic solution.

### 3. Tuberculous Pneumonia

Tuberculous bronchopneumonia or lobar pneumonia frequently results either from a rupture of a tracheobronchial gland into a bronchus or from a severe hemorrhage. In these instances conservative management is indicated at first, the objectives being the relief of pain and cough with medication, and of dyspnea with oxygen. There is disagreement whether a pneumothorax should be induced immediately or whether it is best to wait until the pneumonic process softens. The danger of immediate collapse is due both to the fact that it is at times difficult to collapse a pneumonic lesion and that such collapse precipitates an effusion. I prefer waiting until the inflammatory lesion begins to soften before collapsing the lung, the only exception being in the case of persistent hemorrhage. Another reason for waiting is that not uncommonly the lesion is of the exudative type with no evidence of caseation, and since such inflammations clear with bed rest alone, they should be treated conservatively. In the case of pneumonic lesions the lung should be collapsed as soon as softening takes place.

### 4. Atelectasis

Atelectasis frequently results following a severe hemorrhage or a severe coughing spell. In either case a main or

there exists a pure tuberculous or a mixed infection empyema. All massive effusions, regardless of the complicating bacteriologic organisms, should be removed in sufficient quantity to relieve symptoms, thus permitting visualization of the compressed lung. When definite underlying pulmonary disease exists, it may be advisable to replace the fluid with air and thus establish a therapeutic pneumothorax depending on the condition of the opposite lung. When no evident pulmonary disease is present, the patient should be advised to rest in bed for a period of from three to six months depending on the symptoms in the individual case, regardless of whether tubercle bacilli are found in the pleural fluid.

When the underlying lung lesion is controlled, a tuberculous serous or purulent empyema developing *during the course of pneumothorax therapy* is not so serious a complication as was formerly considered, since spontaneous healing with absorption of the fluid not infrequently takes place with modified bed rest alone. Such an empyema associated with a *bronchopleural fistula*, however, presents a serious complication.

*Mixed infection empyemas* usually result either from infection from without through repeated tappings or from within through the medium of spontaneous or traumatic pneumothorax. This type of complication presents a real problem in management and has an unfavorable influence on the prognosis. Injection into the pleural cavity of a .05 to 1 per cent watery solution of either gentian violet or crystal violet has been helpful in some cases. These solutions have a marked effect on the gram-positive organisms. For the gram-negative ones Dakin's solution or azochloramid, a chlorine compound somewhat weaker than Dakin's solution, has been reported to have beneficial results. Rib resection followed by thoracoplasty occasionally becomes a necessity, especially when a bronchopleural fistula exists. Generally, uncomplicated effusions should be left alone since many heal spontaneously.

## 2. Spontaneous Pneumothorax

Spontaneous pneumothorax may manifest itself as an initial symptom or may appear during the course of the disease.

smaller bronchus becomes obstructed by either blood clots or mucopurulent secretions respectively, thus leading to a localized or generalized atelectasis of either a part of a lobe, an entire lobe, or the whole lung, depending on the size of the bronchus affected. Not infrequently the atelectasis is temporary. In some instances in which the obstruction is the result of stenosis of the bronchi, the atelectasis may be permanent.



Fig. 92.



Fig. 93.

Fig. 92.—Patient G. H. (6/7/41). The pneumothorax was continued for almost ten years. Roentgenogram taken on deep inspiration reveals that the right lung is almost completely re-expanded. Note the accumulation of fluid in the right costophrenic sinus. (See also Figs. 89, 90, 91, 93.)

Fig. 93.—Patient G. H. (6/7/41). Roentgenogram taken on deep expiration reveals the greatest shift of mediastinal structures to the pneumothorax side. Note the deviation of trachea and heart to right. Pneumothorax still present. (See also Figs. 89 to 92.)

*Rolling the patient* from side to side may at times lead to the expulsion of the foreign body if it is a mucous plug. This procedure should not be carried out following a hemorrhage for fear of detaching a blood clot from a bleeding area, thus causing a new hemorrhage. *Pneumothorax* is the treatment of choice provided the contralateral lung permits.

Figures 89 to 93 illustrate the course in a patient who developed a tuberculous pneumonia, plus atelectasis, following

Fig. 89.

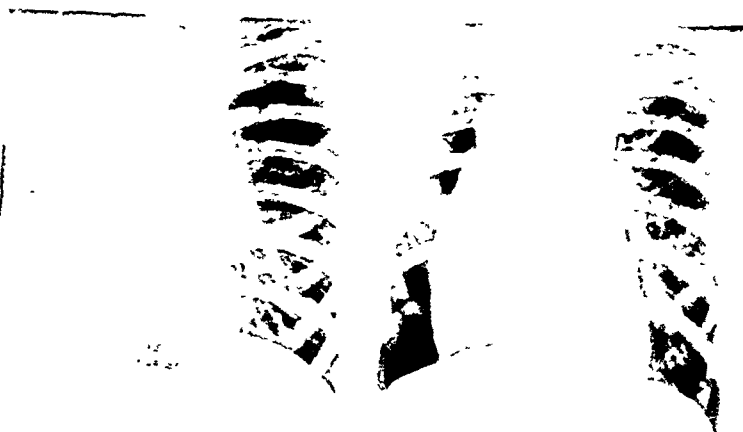


Fig. 90.

Fig. 91.

Fig. 89.—Patient G. H. (9/1/31). Infiltrations and consolidations can be seen in upper half of right lung. Pneumothorax was advised after three months of bed rest, but was refused. Subsequently a pulmonary hemorrhage developed. (See also Figs. 90 to 93.)

Fig. 90.—Patient G. H. (10/29/31). Right-sided atelectasis plus tuberculous pneumonia following the pulmonary hemorrhage. (See also Figs. 89, 91, 92, 93.)

Fig. 91.—Patient G. H. (11/13/31). An artificial pneumothorax has been induced as a therapeutic measure and to stop the hemorrhage. (See also Figs. 89, 90, 92, 93.)

Fig. 94.

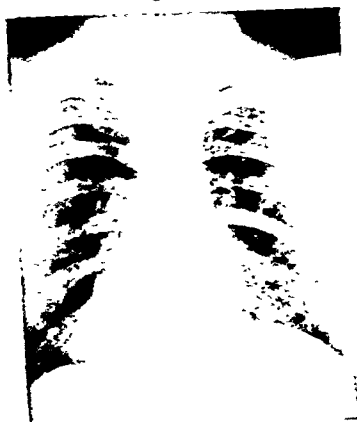


Fig. 95.



Fig. 96.

Fig. 94.—Patient I. L. Numerous calcifications are seen scattered throughout both lungs and especially in right hilar region. A small atelectatic area is present in the right cardiophrenic angle. (See also Figs. 95, 96.)

Fig. 95.—Patient I. L. Pneumothorax on the right side has been instituted. A small atelectatic area is visible at the base during inspiration. (See also Figs. 94, 96.)

Fig. 96.—Patient I. L. Same atelectatic area on deep expiration. Note shifting of mediastinum. (See also Figs. 94, 95.)

the bronchial wall. Mural processes are more frequent, taking the form of a caseous ulcer or of a hyperplastic submucous

severe and repeated hemoptysis. The results of management are shown.

### 5. Tuberculosis of the Tracheobronchial Tree

The frequency of tracheobronchial tuberculosis is not generally known since interest in the subject is relatively recent. The incidence as reported in the literature varies from 3 to 41 per cent. The condition is still frequently undiagnosed and not a few needless thoracoplasties have been done because of a persistent positive sputum which was attributed to active disease in the parenchyma of the lung whereas it had actually originated in a lesion in the bronchial wall.

Probably the chief method of *origin* of this lesion is by direct extension from the pyogenic membrane or cavity along the bronchus. Extension from contiguous lesions in parenchyma or tracheobronchial lymph nodes may also take place. The resulting lesions, regardless of origin, may take a number of *forms*. A diffuse inflammatory change with congestion and edema of the mucous membrane is seen in highly sensitized individuals. This type of involvement usually affects a larger number of bronchi and clears up more readily than other forms. Eloesser<sup>1</sup> describes three types of lesions, namely, the ulcerative, the hyperplastic, and the shrinking, and states that these types may coexist.

The effect of the development of such lesions is to cause either a partial or complete stenosis of the bronchus leading to secondary changes in the lung distal to the obstructed bronchus, and because of this obstructed mechanism certain *symptoms* develop which should be recognized. Dyspnea out of proportion to the amount of pulmonary disease or to the degree of collapse, wheezing, difficulty in raising sputum, or persistent cough with or without large amounts of sputum, should lead one to suspect tracheobronchial tuberculosis. Partial obstruction may lead to localized emphysema and complete obstruction to atelectasis.

Obstruction may be *intramural*, *mural* or *extramural*. The offending tuberculous lesion usually lies in or near the wall rather than in the lumen. In children, especially, a peribronchial or peritracheal lymph node may suppurate and ulcerate

Fig. 94.

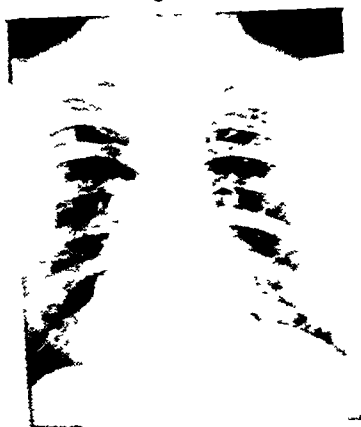


Fig. 95.

Fig. 96.

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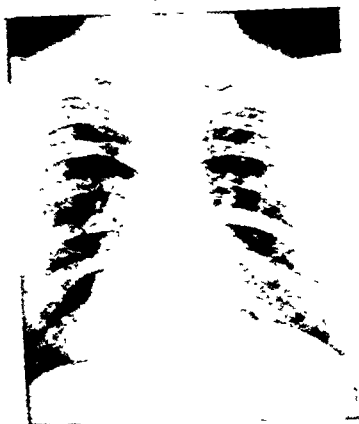


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infection, and physical and roentgen examination of the chest to demonstrate the process in the mediastinum which is giving rise to compression signs. It should also be remembered that enlarged tracheobronchial glands may exist over a prolonged period without producing either signs or symptoms. Figures 97 and 98 illustrate the conditions in two patients.

It is fair to conclude that enlarged tracheobronchial glands are more of a potential danger than an actual one, especially in children. Caseation of the glands with rupture into a bron-



Fig. 97.

Fig. 97.—Mediastinal lymphadenitis. A large node in the left hilar region is visible. The patient was asymptomatic.



Fig. 98.

Fig. 98.—Tracheobronchial lymphadenitis. Large calcified glands are seen in region of superior mediastinum and right hilum. Another film taken four years later showed no change in the glands.

chus may cause hemorrhage and lead to either a tuberculous lobar involvement or a miliary tuberculosis. It is rare to find significant enlargement of the cervical or mediastinal glands as a complication of an active pulmonary tuberculosis, except in first infection tuberculosis, especially in children. The reverse is true in adults, that is, pulmonary involvement more frequently follows primary cervical or mediastinal involvement. More common as a complication of pulmonary involvement is an associated fibrous mediastinitis resulting from a healing or healed tuberculosis.

infiltrate and producing a thickening of the wall of the air passages sufficient at times to obstruct their lumina.

*Treatment* of this condition will depend on the type of involvement of the bronchus, the location, and on the degree of the pulmonary involvement. There is abundant postmortem and clinical evidence to show that the inflammatory lesions and some discrete ulcerations heal spontaneously if the parenchymal lesion is controlled.

For treatment to be effective generally, the obstruction must be relieved. *Bronchoscopic aspiration* of the secretions gives transient relief for dyspnea. *Removal of a tuberculoma* has been successful in relieving symptoms in some cases. *Cauterization* with a 30 per cent solution of silver nitrate has been used by some physicians with reported successes. *Pneumothorax* therapy has been used effectively by the writer, and Figs. 94, 95 and 96, with their explanatory captions, demonstrate the results in one case.

The *actual live wire cautery* has been employed on occasion. *Radiation therapy* weekly for six to eight weeks has been used with reported successes. *Lobectomy* and *pneumonectomy* have also been tried. *Tracheotomy* is at times necessary in order to save life.

In summary it should be stated that this complication is a very grave one, especially when it is associated with marked pulmonary disease. The treatment is generally unsatisfactory insofar as a permanent cure is concerned, and the mortality is relatively high regardless of the type of management. Treatment, however, is helpful in properly selected cases in which the lesion is not too extensive and the pulmonary disease is controlled.

#### 6. Mediastinal, Bronchopulmonary and Cervical Tuberculous Lymphadenitis

Tuberculosis of the mediastinum usually includes enlargement of the tracheobronchial lymph nodes, tuberculous abscess of the mediastinal space, chronic fibrosis of the lymph nodes, together with an associated fibrous mediastinitis resulting from a healing or healed tuberculosis. The diagnosis of tuberculous mediastinitis depends on symptoms and signs of

## 9. Tuberculous Laryngitis

This complication is usually secondary to pulmonary tuberculosis and is rarely if ever primary. It is characterized by infiltrations and the formation of tubercles, granulomas and ulcers in the walls of the larynx, followed later in some patients by edema, fibrosis, perichondritis, chondritis and sometimes necrosis. Statistics of frequency vary from 3 to 97 per cent. The frequency of involvement increases with duration and extent of the disease.

Early tuberculous laryngitis may not present symptoms and may heal spontaneously without recognition. In more advanced cases, symptoms requiring relief are cough, dyspnea, painful and difficult swallowing, and hoarseness. Treatment aims to build up the resistance of the patient, as well as to relieve symptoms, and is accomplished through local measures subordinated to general therapy. The progress of the laryngeal condition following treatment will in general depend on the pulmonary progress.

The treatment can best be carried out in institutions. Early recognition is essential. Prophylactic treatment consists of absolute *voice rest*—with paper and pencil used at all times—and *local applications* in addition to general rest and symptomatic treatment. Intralaryngeal sprays of cocaine, 1 to 20 per cent solution, or chaulmoogra oil, 20 per cent solution, are at times helpful. Topical applications of 2 to 5 per cent solution of formalin and lactic acid have been used. Insufflations of such powders as orthoform and iodoform have been helpful. Inhalations of vapors such as benzoin, menthol and guaiacol have been used. Lozenges such as orthoform and anesthesin are helpful. Heliotherapy and radiotherapy have been tried but are rather dangerous. Cauterization may give immediate temporary and dramatic relief. Blocking of the superior laryngeal nerve relieves pain. Tracheotomy is at times necessary to prolong life. In general, it may be stated that the management of laryngeal tuberculosis where intensive pulmonary disease exists is most unsatisfactory. At the same time one should not assume a fatalistic attitude regarding the incurability of laryngeal tuberculosis in general, since recoveries are frequently seen.

The type of *treatment* employed in tracheobronchial and mediastinal tuberculosis will be governed largely by the presence or absence of caseation of the glands with possible threatening rupture of the capsule, and of pressure symptoms, and by the condition of the lung proper. Bed rest is indicated at all times when symptoms are present, whether or not a pathologic process is present in the lungs. *Mediastinal abscess* resulting from a ruptured tuberculous gland or from a tuberculous vertebra causing pressure symptoms may require surgery.

*Cervical adenitis* has been treated by resection, x-ray therapy, and heliotherapy. The first two procedures are dangerous and the writer has seen miliary tuberculosis develop following this type of management. *Heliotherapy* is probably the best and most effective type of treatment, especially when the pulmonary process has a fibrotic tendency.

#### 7. Cold Abscess of the Chest Wall

Cold abscess of the chest wall may develop spontaneously or after trauma and may heal spontaneously. It may result from a rupture of a mediastinal gland in the anterior mediastinal region causing a mediastinal abscess which may extend to and even perforate the chest wall. It may also develop following repeated punctures for draining a tuberculous empyema. Occasionally cold abscesses open and become secondarily infected. Such abscesses must be drained. Uncomplicated abscesses are best treated conservatively. Heliotherapy, if the lung condition permits, may be tried.

#### 8. Generalized Miliary and Meningeal Tuberculosis

Generalized miliary and meningeal tuberculosis may follow rupture of a caseating gland into a blood vessel. The prognosis of either condition is extremely grave, and the treatment is ineffective. Chronic miliary tuberculosis, which is caused by miliary seeding of a mild infection in an individual with good resistance, explains why occasionally healed cases of miliary tuberculosis are encountered roentgenologically. Recent literature casts a doubt on the existence of chronic miliary tuberculosis and suggests that these cases be considered as types of sarcoid disease.

affected, *renal involvement* is the most important and usually occurs through the hematogenous route. The symptoms requiring treatment are frequent urination, lumbar pain, and hematuria. The treatment is palliative and surgical, depending a great deal on the extent of the pulmonary involvement and on whether one or both kidneys are involved. It should be remembered that some kidney lesions heal spontaneously and for this reason conservatism should be practiced. Surgical intervention is indicated only when the opposite kidney is free from the disease and when the nature of the pulmonary involvement permits it. For *palliation*, rest is by far the most important factor. Heliotherapy has been used when the lung lesion permitted it. Too much light, if not properly administered, may do a great deal of harm.

Tuberculosis of the *epididymis* calls for the same conservatism. Palliative treatment should be tried first, and if this fails, surgery should be considered.

## 12. Tuberculosis of the Skeletal System

Tuberculosis of the vertebral column, especially of the dorsal vertebrae, is occasionally a complication of pulmonary tuberculosis, resulting from a hematogenous dissemination. The management consists of immobilization of the affected part through the medium of splints, braces and plaster casts. Heliotherapy is helpful in many cases. Operative procedures are at times necessary.

## 11. COMPLICATIONS INCIDENTAL TO THE TREATMENT OF TUBERCULOSIS

1. *Spontaneous Pneumothorax and Pleurisy with Effusion.*—Artificial pneumothorax is frequently complicated by spontaneous pneumothorax and pleurisy with effusion. The management of both of these complications has already been discussed. It should be remembered, however, that proper care in the administration of pneumothorax treatment from the viewpoint of technic, frequency of refills and pleural pressure readings will materially reduce the number of spontaneous pneumothoraces, as well as the frequency of pleural effusions.
2. *Air Emboli and Pleural Shock.*—These conditions are at times indistinguishable and occur during the administration of

## 10. Tuberculosis of the Gastro-intestinal Tract

Statistics on the frequency of intestinal tuberculosis vary. Autopsy figures reveal at least 50 per cent involvement of the intestine in pulmonary disease. Undoubtedly, gastro-intestinal involvement exists in many cases without producing symptoms and therefore without recognition. Spontaneous healing occurs in many cases.

The most common site is in the ileocecal region. Slight digestive or abdominal symptoms should suggest the possibility of intestinal tuberculosis. The *symptoms* requiring treatment are pain, alternating constipation and diarrhea, dyspepsia, general abdominal discomfort after eating, and persistent irregular temperature; occasional vomiting may be present.

Protection of the abdomen with an abdominal binder, bed rest and diet will control peristalsis. A *diet* restricting the intake of fluids, peas, beans, cabbage, turnips, potatoes, pastries, rye bread and coarse food in general such as raw fruit and vegetables is helpful. *Heliotherapy* in the form of quartz and mercury lamp, or through the medium of ordinary sunshine, is helpful. Intraperitoneal injection of oxygen has been used.

*Pneumoperitoneum*, 500 to 1200 cc. once a week, has been beneficial in some cases, and aids in checking the diarrhea. Surgical treatment has been tried in the hyperplastic form. *Medical treatment* assumes numerous forms. Calcium chloride, 5 cc. of a 5 per cent solution once or twice a week for four to five doses, helps to control peristalsis and diarrhea. Calcium chloride by mouth is not so effective. Bismuth salts have been widely used. Enemas of warm water with 10 to 20 drops of tincture of opium may help to control severe diarrhea. Opiates may be required. Constipation should be treated with mild laxatives. The ultimate response to treatment will of course depend on the genito-urinary condition.

## 11. Genito-urinary Tuberculosis

The incidence of genito-urinary tuberculosis as a complicating factor varies. In general, it is believed that about 5 per cent of pulmonary cases will show clinical evidence of pulmonary tuberculosis.

While various organs of the genito-urinary tract may be

2. *Mediastinal Displacement*.—This complication frequently follows the re-expansion of a lung following pneumothorax therapy (see Figs. 92 and 93). Aside from occasional dyspnea on effort, symptoms usually are not troublesome. When the displacement of the heart and trachea causes wheezing and marked dyspnea, reestablishment of the pneumothorax, if possible, is at times necessary and sometimes a thoracoplasty is the only procedure that may relieve the symptoms.

3. *Chronic pleurisy*, resulting from healing by fibrosis and thickening of pleura, plus adhesions to diaphragm, is only occasionally troublesome. A chest binder is helpful.

4. *Deformity of the chest wall* with its accompanying atrophy of chest muscles may follow a thoracoplasty operation. Aside from esthetic considerations, this complication is not serious.

#### IV. UNRELATED NONTUBERCULOUS COMPLICATIONS

Tuberculous individuals, like healthy ones, are subject to the development of nontuberculous disease processes. The most frequent is *diabetes* which presents a real problem and makes the prognosis less favorable. The management is of course with diet and insulin. *Hay fever* is common, and if it occurs during the active stage of the disease it can be disturbing.

*Pneumococcus pneumonia* is an occasional complication which is helped by serum plus sulfonamide therapy. *Acute contagious diseases* in the course of tuberculosis are rather rare. *Syphilis* occurs in from 3 to 10 per cent of persons with active tuberculosis in some communities, and should be treated while the patient is in the sanatorium, preferably with small doses of neosalvarsan. *Cancer* and tuberculosis frequently co-exist and treatment consists mainly in relieving symptoms.

#### V. SOCIAL COMPLICATIONS

Whether tuberculous individuals should marry and have children is a debatable question.<sup>2</sup> Marriage and pregnancy during the active stage of tuberculosis do occur despite advice to the contrary by physicians. In the case of pregnancy, abortion should not be attempted. It is surprising, and at times amazing, to find the improvement that sometimes takes

artificial pneumothorax. They are undoubtedly the most dreaded and at times fatal complications incidental to pneumothorax treatment. As soon as these complications are recognized the patient's head should be lowered and stimulants given intravenously. Such a cardiac stimulant as caffeine sodio-benzoate, grains  $7\frac{1}{2}$  is helpful. Injection of adrenalin directly into the heart muscles plus rhythmic artificial respiration may prove successful.

3. *Shifting of Mediastinum and Mediastinal Hernia.*—A hernia of the mediastinum is a protrusion of a pneumothorax sac through the mediastinum into the opposite hemithorax. It is practically always located in the upper anterior mediastinum where the membranes of each pleural cavity lie close together. It is best seen on expiration. The presence of a mobile mediastinum should be recognized early before untoward symptoms arise. High pressures should not be used. Mediastinal hernias will disappear spontaneously as soon as the pleural pressures are reduced.

4. *Other Complications.*—Less frequent and less important complications occurring during the course of treatments are needle track wounds, subcutaneous emphysema, hematoma, pneumoperitoneum and broken needle wounds, and are not of serious import. Rupture of the lung from needle puncture can be serious, but most of these punctures heal quickly. Failure of the lung to re-expand after pneumothorax is discontinued, as seen in Figs. 92 and 93, is at times a problem. Sensitivity to novocain may give rise to alarming symptoms; adrenalin may be helpful in such a case.

### III. COMPLICATIONS INCIDENTAL TO THE HEALING OF TUBERCULOSIS

1. *Emphysema, Bronchitis and Bronchiectasis.*—Compensatory emphysema and bronchitis of the unaffected portions of the lungs occur in nearly all cases of chronic tuberculosis in which there is extensive destruction of the parenchyma. These patients frequently require symptomatic treatment for the cough, which is usually not due to active tuberculosis. Avoidance of upper respiratory infections, climatic therapy when bronchiectasis complicates the picture, and postural drainage are helpful.

## DIAGNOSIS AND TREATMENT OF TUBERCULOSIS IN CHILDREN\*

JEAN V. COOKE, M.D.†

ALTHOUGH in children, as in adults, the most frequent primary localization of tuberculosis is in the lungs, the usual manifestations in children are so different from the disease in adults that, from a clinical standpoint, there are few similarities. In adults, for example, the diagnosis is often suspected because of abnormalities detected in physical examination of the lungs, while in infancy and childhood, only a very small percentage of cases show any early pulmonary changes which can be found by the usual methods of physical examination. In children of all ages, the presence of easily demonstrable pulmonary signs in tuberculous infection usually indicates a late and extensive lesion; and while in adults a localized area of pulmonary dulness or rales is the common finding in early tuberculosis, in older children, such signs are far more likely to be of nontuberculous origin than due to tuberculosis.

*Etiology.*—The initial tuberculous infection in children in almost every instance is the result of *direct contact* with an adult who has an open tuberculous lesion, and the primary portal of entry is the lung parenchyma. While it is possible for infection to arise from ingestion of infected milk with a resulting primary intestinal tuberculosis, such a route is now relatively rare in the United States. This is apparently due to the elimination of the infected animals from dairy herds and to the fact that most of the milk used is pasteurized. From a practical standpoint, tuberculosis in children is to be considered a direct contact infection in which the primary focus is in the lung, while all other routes are unusual.

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place in the lung during pregnancy. After birth the infant should be removed from the environment, and not nursed by the mother. The frequency of relapses occurring after delivery depends mainly on the social and economic condition of the patient.

#### BIBLIOGRAPHY

1. Eloesser, L.: Bronchial Stenosis in Pulmonary Tuberculosis. *Am. Rev. Tuberc.*, 30: 123-180 (Aug.) 1934.
2. Spector, H. I.: Marital Tuberculosis. *Am. Rev. Tuberc.*, 40: 147-156 (Aug.) 1939.

during infancy is well established and this may be attributed to the fact that young children as well as young animals form *antibodies* much less readily than older ones. In tuberculosis the only readily demonstrable antibody is the complement-fixing antibody, and it has been shown<sup>1</sup> that, in infants with tuberculosis, this antibody cannot be demonstrated during the first year of life while from the second to the sixth year gradually increasing numbers of infected children develop it. It seems probable that this inability to form complement-fixing antibodies may be associated with a lack of power to form other protective antibodies and may, therefore, have some relation to the rapid spread of tuberculosis in infancy. The view has been expressed by many that the severity of the disease in infancy is due to greater liability to more intimate contact with an open case and to consequent heavy or repeated infection. This may be a factor, but it would appear more likely that in tuberculosis, as in other infections, infants lack an ability to resist extension of the first infection because of defective immune body formation.

When tubercle bacilli lodge in the pulmonary parenchyma during infancy, a local proliferative lesion begins, and the process develops in one of three ways:

1. *The Proliferative Type*.—The local focus of proliferation may spread with moderate rapidity until the area becomes large enough to be detected by clinical signs or by x-ray. Tubercle bacilli are soon carried to the hilar and posterior mediastinal nodes in which a spreading proliferation and caseation also occur.

2. *The Exudative or Pneumonic Type*.—In some cases the primary proliferative area is accompanied by a pneumonic exudate in the surrounding alveoli which spreads and becomes caseous, often with the later formation of unencapsulated cavities. Here also physical signs of pneumonic consolidation occur and can be demonstrated by x-ray, while the lymph nodes share in the developing lesion.

3. *The Glandular Type*.—In many instances and especially in later infancy the primary proliferative process in the pulmonary parenchyma remains relatively small and does not spread to a degree sufficient to give physical signs, or even to be demonstrable by x-ray. From it, however, the organisms are carried to the lymph nodes at the hilum and especially to those in the posterior superior mediastinum, and here the tuberculous process develops to a degree which overshadows the lesion in the parenchyma. In many cases this glandular tuberculosis represents the chief or only lesion seen in x-ray (Fig. 100). It is, of course,

*Classification.*—The concept that tuberculous disease shows striking differences in its development and manifestations between the first or initial infection, and a later reinfection appears well established. In adults the usual type of phthisis is of the characteristic reinfection variety and at times older children may develop this reinfection or adult type of disease. Such cases, however, are relatively infrequent and in the majority of instances the disease in children is of the *first infection type*. In such initial infections the most important consideration which influences the character of the lesion produced is the *age* of the child when the infection is acquired. In infancy the infection tends to be an acute and serious rapidly spreading disease, while in later childhood the tuberculosis which follows the initial infection is usually much less severe and is likely to remain localized.

For clinical and descriptive purposes, tuberculosis in children may be classified as: (1) infantile tuberculosis; (2) juvenile infection; (3) adult type, or reinfection tuberculosis.

### INFANTILE TUBERCULOSIS

#### Pathogenesis

The characteristic feature of the infantile type is the relatively *rapid spread* of the disease. This is apparently associated with a *lack of resistance* to the extension of the infectious process and is quite similar to the manner in which tuberculosis spreads in the guinea pig after experimental inoculation. In this animal there appears to be no mechanism to arrest the continued and relatively rapid extension of tuberculous infection and within a few weeks the disease has produced very extensive lesions. This is also true in most infections in infants so that the usual picture tends to be that of an acute rapidly progressive disease. This lack of resistance to the spread of first infection persists through infancy but becomes less striking by the middle of the second year. First infections acquired after infancy usually do not exhibit this unrestrained extension in the pulmonary parenchyma, so that in later childhood the body acquires an ability to inhibit the spread of the tuberculosis. This change is apparently not associated with the development of skin sensitivity, since a positive tuberculin skin test is found with the same regularity in children of all ages after infection.

In general, the greater severity of almost all acute infections

four days, and all showed very extensive generalized tuberculosis at necropsy. The intradermal tuberculin test was negative in one baby on the twentieth day (two weeks before death) while another showed a positive test on the twenty-fourth day (eight days before death).

### Clinical Manifestations

The symptoms of infantile tuberculosis in its earlier stages are so few and slight that there is no suspicion of illness. Indeed, it is not uncommon to observe a baby with extensive generalized tuberculosis in whom the symptoms are so mild that the serious character of the disease would never be suspected from them. As the early infantile disease progresses, however, certain features usually become apparent. The most frequent is *failure to gain weight*, although there is usually little tendency to such gastro-intestinal disturbances as vomiting or diarrhea. *Fever* is often present, although it may be slight. It often varies from  $99^{\circ}$  to  $102^{\circ}$  F. ( $37.2^{\circ}$  to  $38.9^{\circ}$  C.). Most patients do not appear toxic, while cough and respiratory symptoms are usually absent unless there is some superimposed secondary infection. Eventually, however, with further extension of the lesion, signs of real illness tend to appear, of which *fretfulness*, irregular fever and often a *cough* which may assume a "brassy" character, may be prominent. Evidence of pulmonary consolidation may be found in the proliferative or in the pneumonic types with localized rales, while in the glandular type such signs may be absent. The spleen is often palpable. Even in generalized infections few local clinical signs are apparent unless tuberculous meningitis develops.

### Diagnosis

The diagnosis of infantile tuberculosis is almost entirely dependent upon the use of the tuberculin skin test and x-ray of the chest. The possibility of infection should always be considered when any constitutional symptoms such as fretfulness or fever persist and are unexplained, when an infant fails to gain, or when there is any history of contact with known tuberculosis. Those instances also, in which physical signs of pulmonary consolidation with localized rales in an infant are

true that in cases of this type which come to necropsy, the original focus in the pulmonary parenchyma is almost always demonstrable after careful search even though the disease is much more extensive in the lymph nodes.

It must be understood that the foregoing grouping is made for descriptive purposes and chiefly to serve as a basis for discussion of the clinical types. Practically, although the proliferative or pneumonic or glandular element may dominate the picture in any case, the processes overlap to a considerable degree. In any instance in which the pulmonary disease involves the pleura, a pleurisy results, but this is often not detected clinically. Usually only a small amount of fluid exudes, which gives few clinical signs, and only occasionally in older infants does a massive exudate occur. Such fluid is yellow, shows a predominance of lymphocytes, and soon clots, differing from tuberculous pleural exudates in adults only in having a somewhat higher cell content and more polymorphonuclear elements. Even in later childhood the formation of massive pleural exudates so common in adults, is relatively infrequent. In any type of infantile tuberculosis it is obvious that the lesions must reach a certain size before being demonstrable, and the earliest symptoms of disease may, therefore, be more difficult to recognize.

While early development of the infection may follow one of the courses outlined, unless arrested all tend to progress and to terminate in a generalized miliary tuberculosis. The dissemination of the disease always results from the secondary lymph node involvement at the pulmonary hilum. A softened node erodes a small vein with the production of generalized tuberculosis, or, in some instances, the erosion of a bronchus produces an extensive pulmonary dissemination or a rapidly spreading tuberculous pneumonia. In practically all fatal cases of tuberculosis during infancy the disease has become generalized.

*Congenital Tuberculosis and Tuberculosis of the Newly Born.*—While infection may occur *in utero*, such instances appear to be relatively rare, but the newly born are especially susceptible, and rapidly fatal infections acquired in the early days of life are not uncommon. Apparently any baby born of a mother with an open tuberculosis is doomed to an early death unless removed from contact immediately after birth. In three recently observed cases in which the infection was so acquired, the infants lived twenty-five, thirty-two and thirty-

nothing about the localization of the infection, the frequency of a pulmonary focus makes it necessary to have a chest roentgenogram on all patients with positive reactions. The variation in the picture is considerable, but usually the lesion revealed is more extensive than is suspected from the clinical signs. It must be remembered also that the x-ray shows only areas which have reached a certain size and that other early



Fig. 100.—Infantile Tuberculosis. Mediastinal type of glandular tuberculosis in a two-year-old child resembling thymic shadow.

developing foci may be present but unrecognizable on the film.

A typical film of a fifteen-month-old baby with *proliferative-pneumonic type* of tuberculosis in the left upper lobe is shown in Fig. 99. The appearance here is not characteristic of tuberculosis and is quite indistinguishable from that seen in an acute pneumonia. The identification of its tuberculous character must be made by the absence of accompanying signs and symptoms of acute pneumonia and by the presence of a positive tuberculin test and possibly other previously mentioned clinical manifestations suggestive of tuberculous disease. Not all patients show such a clear-cut outline of con-

not accompanied by acute pneumonic symptoms, should be suspected.

*Tuberculin Test.*—The most reliable test for clinical use is the intradermal injection of 0.1 cc. of a 1:1000 dilution of tuberculin (O.T.) which should be read after forty-eight hours. The patch test, which consists of the application of tuberculin by means of impregnated adhesive, is only slightly less reliable. The importance of the tuberculin test during infancy is that a properly carried out negative test is quite

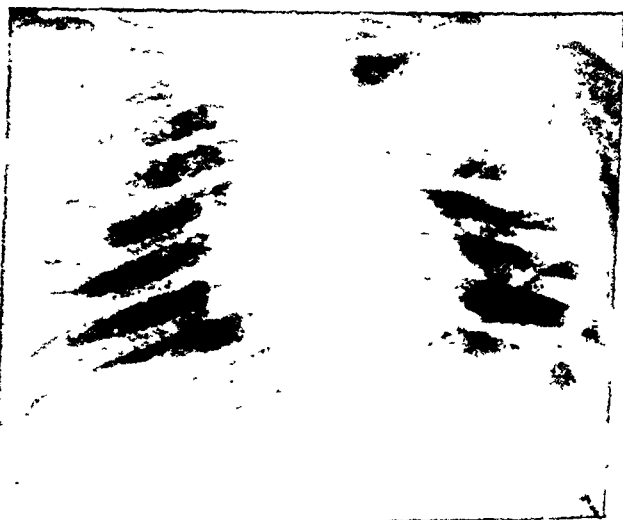


Fig. 99.—Infantile Tuberculosis. A fifteen-month-old baby with pneumonic type of tuberculous pulmonary involvement.

trustworthy in excluding tuberculosis since it is only in very exceptional instances that infected children have negative reactions. On the other hand, a positive tuberculin reaction in itself is usually interpreted to mean only the presence of infection which may be either mild and latent or active disease. During infancy, however, the large majority of babies with positive tuberculin tests have active infections and a positive reaction must therefore be considered of much more serious import than in an older child.

*Chest X-ray.*—Since a positive tuberculin test indicates

gests such a type of tuberculosis previously. Four children between the ages of eight and thirteen years have been seen in recent years at the St. Louis Children's Hospital with such calcified foci. None had any clinical evidence or history of tuberculous infection and only one had a positive tuberculin skin test. A chest roentgenogram (Fig. 101) of the youngest child in this group shows the characteristic, scattered, calcified nodules. There is a growing belief that such cases are not

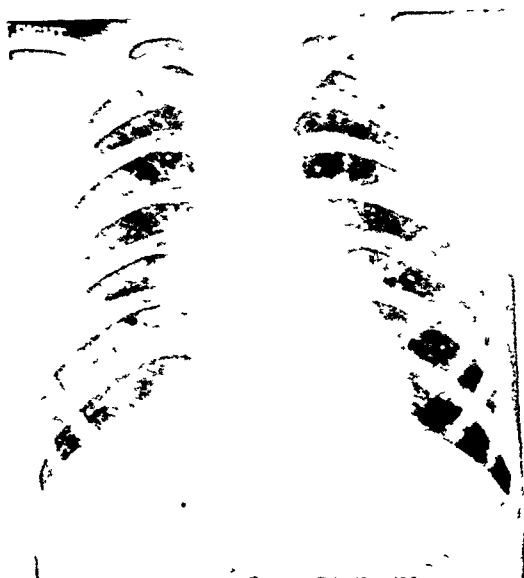


Fig. 101.—“Healed Miliary Tuberculosis.” Multiple calcified foci with some hilar thickening in a healthy eight-year-old boy with negative tuberculin reaction.

of tuberculous origin but are due to some other cause. One of the suggested explanations, which is supported by considerable evidence,<sup>2</sup> is that they result from a low grade fungus infection with one of the aspergillus group.

*Demonstration of Tubercle Bacilli in Stomach Washings.*—

It is usually so difficult to obtain sputum in babies that search for tubercle bacilli in gastric contents after fasting is more practical. The washings after lavage with about 100 cc. of water are treated with sodium hydroxide to dissolve the

solidation and at times an irregular patchy shadow may be seen, although almost always the picture suggests an area of pneumonia.

The *glandular type* of infantile tuberculosis gives a much more characteristic picture. Here the shadow is above the heart and appears to be a widening of the mediastinum as in Fig. 100. This might easily be mistaken for a thymic shadow except that during later infancy, at which time this glandular form is somewhat more common, the thymus is rarely enlarged. The infected and visibly enlarged nodes are in the superior mediastinum. Although those nodes lower at the pulmonary hilum are usually also involved in the infection, they are much less frequently visible in the x-ray during infancy than in later childhood. Such a widened mediastinal shadow in a chest film should arouse the suspicion of tuberculosis which may have been previously unsuspected. Its diagnostic significance has not been emphasized as much as it deserves.

*Generalized Miliary Tuberculosis.*—The certain diagnosis of generalized tuberculosis can be made only in its later stages when the pulmonary foci have become grossly visible and give the characteristic and easily recognized "snowstorm" picture in a chest film. Since no special clinical manifestations accompany the general dissemination of the disease other than may be found in severe localized tuberculosis, the presence of a previously unsuspected miliary tuberculosis may be revealed on x-ray examination. It should also be remembered that even in such films as Figs. 99 and 100, there may be disseminated pulmonary tubercles too small to be seen. The presence of clinical signs of tuberculous meningitis in an infant is almost always an indication of disseminated tuberculosis and in a majority of the fatalities in infantile tuberculosis miliary tuberculosis is found at necropsy.

*"Healed Miliary Tuberculosis."*—Disseminated or miliary tuberculosis represents such an extensive type of infection that recovery is usually believed impossible. There are, however, curious instances both in children and adults in which the x-ray shows many disseminated calcified nodules scattered throughout the lung parenchyma. These are usually found in persons without symptoms and with no history which sug-

### Therapy

Probably the first consideration in therapy is the *prophylaxis* of infection. The greatest care should be taken to protect infants from contact with any person with known tuberculosis, for reasons previously emphasized. This means not only the members of the household but includes nurses and servants. Whenever a tuberculous infection is found in any child, an immediate and careful survey of the familial contacts should be made to determine if possible the source of the disease. It is only by such a search that the individual responsible may be detected and further spread of infection to the patient and possibly to other children prevented.

After infection has occurred, the treatment is general and symptomatic. Most important is the *nursing*, with special attention to nutrition. The diet should be adequate and include all necessary vitamins. Care must be taken to prevent exposure to all infections of the upper respiratory tract, and especially measles and whooping cough. Most young children with evidence of clinical activity can be better treated in a hospital until there is an apparent arrest of active disease.

### JUVENILE TUBERCULOSIS

#### Pathogenesis

First tuberculous infections after infancy tend to be of a much milder type than in early life. Mention has been made of the general influence of age on the development of pulmonary tuberculosis in children and that in early infancy there is a tendency to rapid spread in the lung parenchyma while in older infants the tuberculous process develops and extends in the bronchial and mediastinal lymph nodes more readily than in the lung itself. In older children the primary focus in the parenchyma has still less propensity for developing the type of proliferative disease common in infantile tuberculosis but usually produces a relatively small and insignificant lesion. The organisms, however, are carried by the lymphatics to the pulmonary hilum and posterior mediastinum which become enlarged from a proliferative tuberculosis. In many such cases followed clinically and by pulmonary x-ray studies, the absence of demonstrable pulmonary foci is rather remarkable. At necropsy, also, in children who have died from other causes, and in whom these mediastinal glands are found tuberculous, the primary lesion in the lung, if found at

mucus, centrifuged at high speed, and the sediment stained for acid-fast organisms. Their presence makes the diagnosis absolute, although in most instances in which organisms are demonstrable by this method, the disease has progressed to a fairly advanced stage.

### Prognosis

The prognosis in infantile tuberculosis is dependent on several factors of which the most important are: (1) the *age* of the patient, (2) the probability of *continued contact* with an infected adult, (3) the *extent and severity* of the lesion, (4) the *manner in which the child is reacting* to the infection, (5) and the care and attention to proper nursing and nutritional requirements.

In earlier infancy the infection is usually more severe and rapidly progressive than later, and it is obvious that those patients who are not removed from contact with the source from which their infection arose will continue to receive organisms. Because the early symptoms of infection are so indefinite, many patients are not seen until the lesions are well advanced, and in such cases the outlook is, of course, serious. Probably one of the most important features in prognosis is the way in which the infant reacts clinically to the infection. If the disease appears to be progressive with continued fever, lack of weight gain, and other evidence of toxicity, the prognosis is grave. On the other hand, when such signs are absent and the patient is free from fever and gaining weight, the outlook for an arrest of the infection is much better.

In general, the prognosis of infantile tuberculosis when the lesions have become manifest and easily demonstrable is very serious and the disease tends to progress with moderate rapidity. Not all infants with tuberculous infection progress in this manner and even gross lesions easily demonstrable by x-ray may become arrested and recover. Such an outcome is more frequent in earlier, milder infections. In any individual case the prognosis rests on the factors mentioned and possibly to differences in individual resistance. Usually no opinion about the outcome is possible until after a varying period of observation.

suspicion. Certain symptoms such as *cough* and *night sweats* are often present in early tuberculosis in adults, but the error should not be made of considering such symptoms as common manifestations of early tuberculosis in children. While occasionally enlarged tracheobronchial nodes may cause cough in a child, this is of a fairly distinctive "brassy" character, and apparently due to pressure, while a "bronchial cough" in a child is rarely of tuberculous origin. Night sweats also are not especially uncommon in childhood and may be due to a variety of causes. They are of little significance so far as juvenile tuberculosis is concerned.

On *physical examination*, the findings are often equally indefinite. While severe malnutrition is unusual, most children are on the lower limits of average nutrition for their age and height, and have a mild degree of *anemia*. The enlarged mediastinal lymph nodes usually give no physical signs, although a very expert observer may sometimes detect slight increase in *paravertebral dullness* and a positive *D'Espine's sign* (whispered pectoriloquy extending below the second dorsal spine). While the presence of *phlyctenular conjunctivitis* makes the diagnosis of tuberculosis certain, such children tend to show no more demonstrable evidence of pulmonary disease than those without it, and this is true also of the occasional child with skin *tuberculids* of the papulonecrotic or lichen scrofulosorum type. The skin tuberculin test is positive and the x-ray shows pulmonary changes which will be mentioned later.

In those children in whom the process has spread beyond the usual type of moderate mediastinal proliferation usually present, the chief added symptoms are *irregular fever* and possibly an increase in *asthenia*. As a rule, no additional physical signs of pulmonary disease are present except in the occasional instance of pleural effusion. Even in very severe spreading or early generalized infections, the pulmonary physical signs are minimal and usually not detectable by ordinary examination. Lesions from extrapulmonary metastatic foci in such locations as the bones, peritoneum or nervous system usually present obvious clinical signs.

all, is usually small and well localized. Sometimes only a small calcified nodule remains of this initial "Ghon" lesion. The course of the infection in this juvenile type of tuberculous infection is usually benign, and it remains well localized in the affected lymph nodes. In this respect there is a *much greater resistance* to the spread of the disease than during infancy.

The development of the lesion in the mediastinal lymph nodes may follow several courses. In the majority of cases there is proliferation of tuberculous tissue with some caseation which remains localized and encapsulated with later fibrosis or calcification. Occasionally there may be softening of a gland and a few tubercle bacilli may reach the blood stream, possibly through the lymphatics, and lodge in various parts of the body. Most of such *metastatic lesions* probably heal without clinical manifestations and may be accidentally found many years later at necropsy as small fibrous foci. In certain instances, however, some of these organisms may lodge in the growing bony epiphyses with resulting tuberculous spine or hip, or accidental localization in the nervous system may result in tuberculous meningitis. The glandular lesion also may occasionally become softened, and rupture into a vein with a resultant disseminated tuberculosis in a manner similar to the infantile type, or involve an adjacent portion of the pleura with the production of pleurisy with effusion. Occasionally the nodes may show a massive enlargement with tumor-like extension out into the lung fields (Fig. 105). It is to be emphasized, however, that the characteristic features of juvenile tuberculous first infections are referable to the *lymph nodes* and not to the pulmonary parenchyma, that most of such infections are relatively *benign and localized*, and that an *extension* of the disease may occur in certain children.

### Clinical Manifestations

The symptoms produced by juvenile tuberculosis are notoriously mild, inconstant and variable. One can only enumerate certain ones which may suggest the infection. A history of *frequent coughs and colds* is common, and the complaint of *tiring easily* with some disinclination for physical exercise. *Appetite* may be poor and the *weight* stationary, although any striking loss of weight is somewhat unusual. Transient periods of *slight, unexplained fever* may be noted, sometimes with afternoon elevations. A history of known exposure to tuberculosis is inconstant but when present should, of course, excite

with far greater frequency than those on the left. This is fortunate for radiologic observation since the left hilum is usually concealed by the heart shadow. The typical picture of juvenile tuberculosis is shown in the roentgenogram of Fig. 102, in which a fairly sharply outlined shadow is seen at the right hilum just lateral to the heart. The chief characteristics are the location of the shadow just to the right of the heart, often with a rounded, crescentic or triangular shape, and the fairly uniform density, although the central portion may be slightly denser than the periphery. While the streaking of increased bronchial tree marking may be present in some degree, this is apparently not a part of the tuberculous process and is usually not a prominent feature.

Often the shadow of the enlarged tuberculous nodes is not continuous with the cardiac shadow, but separated from it by a straight or slightly curved, clear, air-containing linear streak of varying width as seen in Fig. 103. In the first x-ray (Fig. 102) is seen the type of shadow to be expected in an early juvenile infection without striking clinical signs, although the shadow is often smaller in size. The second (Fig. 103), however, shows a much more extensive lesion since in addition to the hilar shadow, there is a large tumefaction in the superior mediastinum above the heart. Such a picture in itself suggests a rather alarming degree of activity, and represents one type of spread of juvenile tuberculosis. In neither is there evident any primary focus in the lung parenchyma, and it is only in occasional instances that such an area is apparent in the film.

*Nontuberculous infiltrations* of the pulmonary hilum are seen with moderate frequency in children and are usually the result of recurring respiratory infections. Such children often have infected paranasal sinuses and tonsils in which acute infection arises and spreads to the bronchi so that they suffer from subacute recurring bronchitis. The x-ray picture of such hilar infiltrations has characteristics distinct from those seen in juvenile tuberculosis, and it can usually be distinguished without much difficulty. A typical example of such nontuberculous infiltration is shown in Fig. 104, in which rather coarse bronchial markings converge at the right hilum to pro-

## Diagnosis

As in the infantile type, the use of the *tuberculin skin test* and chest roentgenogram are the most valuable methods in the diagnosis of juvenile tuberculosis and constitute a most important addition to the rest of the examination. In every case it is usually necessary to review all the data available in order to form a reliable opinion about the extent and severity of the disease. Since there is no single criterion for the degree of activity in the infection, a positive tuberculin skin test is a

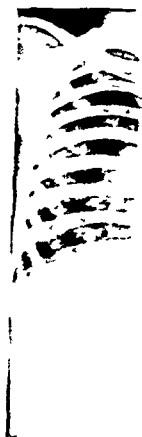


Fig. 102.



Fig. 103.

Fig. 102.—Juvenile Tuberculosis. Typical well outlined shadow at right hilum in six-year-old boy.

Fig. 103.—Juvenile Tuberculosis. Sharply defined right hilar shadow characteristic of tuberculosis in a ten-year-old child. Tuberculous "mediastinal tumor" also present.

less certain indication of an active infection in older children than during infancy, but it must be remembered that activity cannot always be excluded by the absence of clinical signs of disease.

*X-ray of the chest* in children with the juvenile type of tuberculosis usually shows a rather characteristic shadow at the right hilum. Although the reason is not well understood. Still<sup>3</sup> and others have observed that the lymph nodes on the right side are involved by the disease more extensively and

Here is seen the site of the original primary pulmonary infection as a small calcified nodule in the right lower lobe. In other instances a spread to the pulmonary parenchyma may be evidenced by an unexpected triangular shadow extending out from the hilum. In many instances the slightly or moderately enlarged hilar lymph nodes regress after a varying period, and the only evidence seen in the roentgenogram is an irregular calcification in this area. Such calcification almost always represents an inactive healing lesion.

### Therapy

An older child with a positive tuberculin skin test and an x-ray showing some evidence of the hilum infiltration accompanying juvenile tuberculosis, but without clinical signs of disease, is frequently spoken of as having only a "tuberculous infection" which is thought of little importance. Such a child is considered to be in a different category from children with manifest signs of activity who have "tuberculous disease." While it is true that most children with the juvenile type of tuberculosis tend to recover without serious extension, it must be remembered that those cases in which the disease spreads and becomes manifestly active, have originated in such masked juvenile infections. This indicates the necessity for *early recognition* of the juvenile type of tuberculosis and for adopting a regimen which will minimize the danger of its spread. It would seem, therefore, that "tuberculous infection" in children should probably be regarded of sufficiently serious potentialities to warrant somewhat more consideration than it is usually given, even though the actual percentage of children who develop a spread of the disease is small.

The management of a child in whom the diagnosis of juvenile tuberculosis has been made will depend upon the evidence of *activity* indicated by a review of the symptoms and the x-ray changes present. Sometimes a period of observation during which temperature readings were recorded is desirable.

*Symptomless Cases.*—If there are no symptoms, if normal activity does not produce fatigue, temperature remains below 99.5° F., the child is alert with good appetite and gaining normally in weight, and if a positive skin test with some cal-

duce a shadow not sharply outlined and with irregular density tending to extend along the bronchi into the lung fields. It lacks the somewhat circumscribed character seen in the tuberculous infection.

It is, of course, not possible, or to be expected that one can identify with certainty all juvenile tuberculous infections from an examination of the x-ray alone, and the diagnosis in every case must be made by a review of all available data. It is of interest, however, that from the characteristics out-



Fig. 104.

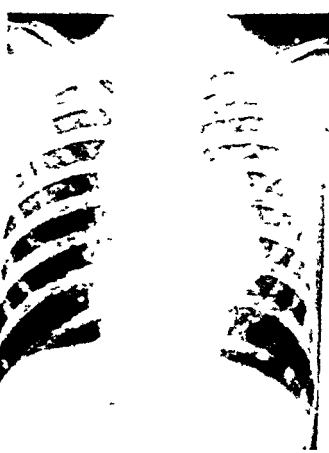


Fig. 105.

Fig. 104.—Typical *nontuberculous* pulmonary hilar shadows from recurring respiratory infections in a six and one-half-year-old child. "Sinus lung."

Fig. 105.—Juvenile Tuberculosis. Extensive spreading active glandular tuberculosis resembling Hodgkin's disease in an eleven-year-old girl. Calcified Ghon tubercle in right lower lobe.

lined, one can after a moderate experience predict with considerable accuracy from an examination of the chest x-ray alone those children who will have positive tuberculin skin tests.

One type of extension or active spread of juvenile tuberculous infection to the superior mediastinal nodes has been illustrated in Fig. 103. In other instances there may develop a rather massive enlargement of the glands extending out into the lung fields and mediastinum as that shown in Fig. 105.

quate, well balanced, high caloric *diet* in which all necessary vitamins are included; suitable exposure to *fresh air* and judicious use of *heliotherapy* with carefully graduated exposure to sunlight; the prevention of exposure to acute respiratory infections; and such local or symptomatic treatment as may be indicated in the individual child. Continued clinical observation of the patient's response to the infection together with occasional x-ray check on the progress of the lesion should be carried out until the symptoms disappear and the disease is considered in an inactive stage. This may require a number of months.

With the gradual resumption of normal activity the same precautions previously suggested in the management of initial juvenile infections should be observed. As a rule, drugs play little part in the recovery from tuberculosis, and even the newer sulfonamide products have not been shown to have any definite effect. Drug medication should be used only for specific indications.

*Tuberculin in Treatment of Phlyctenular Keratoconjunctivitis.*—Specific therapy with tuberculin has been unsatisfactory and disappointing except in the treatment of one of the more common manifestations of childhood tuberculosis—phlyctenular keratoconjunctivitis. Such "desensitization" with tuberculin is apparently the only form of therapy with any constant beneficial effect on this condition. Subcutaneous injections are given once or twice weekly starting with 0.1 cc. of 1:1,000,000 dilution of Old Tuberculin. The dose is gradually increased, usually by doubling the preceding dose, until 0.5 cc. of 1:1000 dilution can be given without signs of local reaction. If such local reaction results from any injection, a reduction of the amount given and more cautious increase is indicated. Clinical improvement is slow and even after recovery there is a tendency for recurrence unless the treatment is supplemented by general hygienic and dietary measures for the relief of the underlying infection.

#### ADULT TYPE OF TUBERCULOSIS IN CHILDHOOD

Children who have previously had an initial infection of the juvenile type of tuberculosis may later become infected again

cification in the hilar lymph nodes is the only radiologic evidence of an apparently old or latent infection, then no restriction of his normal activities is necessary. He should be examined from time to time to facilitate the early recognition of any unexpected tuberculous activity, while nutrition should be maintained and regular temperate habits should be encouraged.

*Subclinical Infection.*—A child in whom easy fatigability, even moderate malnutrition with anorexia, occasional slight fever, and any symptom suggesting a subacute infection, are present, should occasion more concern. This is especially true when a positive tuberculin test is accompanied by a chest x-ray in which the hilar nodes are sharply visible, moderately enlarged and have the characteristics previously mentioned as often associated with recent infection and subclinical activity.

In such instances, somewhat more careful supervision is indicated. As a rule such children need not be kept from school unless the asthenia is striking, since they are not likely to transmit the infection. Exercise and activity should be restricted to that amount which is well tolerated. Respiratory infection should be prevented insofar as possible by adequate clothing and by guarding against chilling from rain and exposure. Regular habits of eating and sleeping are important and a nutritious, high caloric diet is advisable. Sometimes the appetite may be improved by the administration of vitamin B factors. Morning and evening temperatures should be followed, and the development of fever should indicate rest in bed until it subsides. Regular observation of the clinical status and of pulmonary x-ray changes should be continued on such children until the manifestations of even "subclinical" activity disappear. As the child improves, appropriate relaxation of previous restrictions may be allowed with careful check of their effect on the child's progress.

*Manifest Tuberculous Disease.*—Little need be said regarding the management of children in whom tuberculous disease has become manifest, since the principles of therapy are those now generally employed in all active tuberculosis. These include complete *bed rest* so long as fever is present; an ade-

## DIAGNOSIS AND TREATMENT OF EXTRAPULMONARY TUBERCULOSIS\*

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THE lungs constitute the most frequent locus of the primary tuberculous infection and are also by far the most common site of progressive tuberculous disease. It must be constantly remembered, however, that tuberculosis is a systemic infection and that extrapulmonary manifestations either may dominate the clinical picture or complicate the course and management of pulmonary lesions.

Extrapulmonary tuberculous processes may be conveniently separated into *three groups*: (1) disease initiated by surface contamination and inoculation of mucous surfaces with bacillus-laden material from an infected lung; (2) lesions resultant from the lymphogenous or hematogenous dissemination of organisms from the primary site of invasion; and (3) spread of infection to contiguous structures.

It will be noted that this classification neglects the relatively rare instances of primary extrapulmonary tuberculosis, and further overlooks the fact that any of the enumerated mechanisms may be capable of inducing tuberculous involvement of a given organ. Differentiation into these groups, however, serves to emphasize certain practical *clinical considerations*. In the presence of a demonstrably active pulmonary tuberculosis, special attention is naturally given in the history and examination to the possible existence of lesions in other parts of the body; group 1 lesions predominate in this situation and should be sought for with particular diligence. When the physician encounters disease located in an organ in which tuberculous lesions are produced by the group 1 mechanism,

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from contact with an active case. This *reinfection* produces a pathologic and clinical picture entirely different from the first infection. The allergic sensitivity to tuberculin which develops soon after the initial infection, and which is manifest by the positive tuberculin skin test, involves all the body tissues and changes their reactivity to the tubercle bacilli and its products. Consequently, when some organisms lodge in the lung of an individual sensitized by a previous infection, a local proliferative reaction in the pulmonary parenchyma develops. This differs from that seen in the spreading infantile type in being slower in developing and in having a greater tendency to be walled off and to remain circumscribed by the connective tissue proliferation. Some of the infecting organisms are carried to the lymph nodes at the root of the lung, but the glandular manifestations are secondary and overshadowed by those of the process in the parenchyma. This lesion as it develops produces local signs of rales and impaired resonance together with constitutional symptoms of fever and asthenia.

The clinical and pathologic picture in children with the reinfection type of tuberculosis as well as the treatment is identical with phthisis in adults. It is essentially a disease of later childhood and only occasionally encountered in younger children. It is believed that in some instances such reinfection tuberculosis may result from an autogenous spread from an activated initial lesion rather than from an outside source. The extent of the pulmonary involvement as shown in the x-ray is often much greater than anticipated from the physical signs.

#### BIBLIOGRAPHY

1. Cooke, J. V.: Complement Fixation for Tuberculosis in Children. *Am. J. Dis. Child.*, 21: 78, 1921.
2. Sayers, R. R. and Meriwether, F. V.: Miliary Lung Disease Due to Unknown Cause. *Am. J. Roentgenol.*, 27: 337, 1932.
3. Still, G. F.: *Common Disorders and Diseases of Childhood*. Ed. 5. Oxford University Press, London, 1927, p. 486.

*spasm* are sometimes evident in the right lower quadrant. Final diagnosis usually must be established by roentgenologic study, utilizing the technic of Brown and Sampson.

*Differential Diagnosis.*—The signs may closely simulate those of *appendicular disease*. Whereas the common error is the making of an improper diagnosis of appendicitis in an individual whose symptoms are the consequence of tuberculous bowel ulcerations, it is a fact that tuberculous patients are not exempt from acute gangrenous appendicitis. When abdominal pain is acute in onset, accompanied by vomiting, tenderness, and muscle guard localized in the right lower quadrant, laparotomy should be performed.

A roentgen film of the chest is helpful in distinguishing intestinal tuberculosis from intra-abdominal disease of other etiology. If radiologic evidence of lung disease is lacking, tuberculosis can be excluded with fair assurance. A careful roentgen survey of the gastro-intestinal tract, proctoscopy, and bacteriologic study of the stool should be carried out whenever the diagnosis is in doubt.

*Treatment.*—Patients with the ulcerative form of enterocolitis should remain at *bed rest*. When the involvement is not extensive, improvement of the intestinal lesions will often progress hand in hand with the healing of the pulmonary disease. Decrease in the amount of sputum and the disappearance of bacilli from bronchopulmonary secretions are of considerable importance since the swallowed sputum aggravates existing intestinal lesions. The *diet* must be soft and low in residue. All seeds, skins and strings should be eliminated. This may be accomplished by requiring that vegetables be pureed and that fruits be limited to the strained juices or stewed fruits. Fat meats and whole grain breads are usually interdicted. It must be emphasized that a low residue diet is ordinarily deficient in vitamins. Addition of *brewer's yeast* or one of the concentrates is therefore desirable to maintain an adequate supply of the vitamin B complex. The *cod liver oil* factors are of particular importance and may be supplied in a mixture prepared by floating one to two tablespoonfuls of oil on the surface of 3 ounces of orange or tomato juice, and administered after each meal. If an idiosyncrasy or marked

it is imperative that a careful roentgenologic investigation of the chest and microscopic examination of the sputum be performed. If the lungs are uninvolved, the local organ disease is almost certainly nontuberculous. In contrast, groups 2 and 3 processes may occur with or without active pulmonary tuberculosis.

#### 1. COMPLICATIONS ORDINARILY INITIATED BY INOCULATION OF MUCOUS MEMBRANE BY SPUTUM

##### Gastro-intestinal Tuberculosis

Although tuberculosis may involve any portion of the alimentary tract, the ileocecal and anorectal regions are the only areas affected with sufficient frequency to warrant extended discussion.

**ILEOCECAL TUBERCULOSIS.**—Intestinal tuberculosis constitutes the most frequent complication of the pulmonary disease, occurring in the majority of all patients dying as a result of tuberculosis. The tubercle bacillus like the bacillus typhosus exhibits a predilection for the lymphoid tissue of the terminal ileum and cecum. The early lesions take the form of mucosal and submucosal infiltrates, and are followed by the appearance of ulcerations which extend in girdle fashion perpendicular to the long axis of the bowel.

**Clinical Picture.**—Diagnostic problems are presented by the occasional occurrence of extensive tuberculous enteritis without symptoms referable to the intestinal tract, and conversely by the distressing functional derangements of the digestive apparatus which so often accompany the toxemia of isolated pulmonary disease. *Abdominal pain* and *diarrhea*, when present, suggest ileocecal tuberculosis, but absence of these symptoms does not exclude organic intestinal involvement. *Demonstration of blood in the stool* is strong confirmatory evidence of intestinal tuberculosis. In general, one should suspect the possibility of ileocecal disease in any patient with pulmonary tuberculosis who manifests abdominal symptomatology and in any person with active pulmonary involvement who exhibits fever, weight loss, and unfavorable progress which appear out of proportion to the severity of the lung lesion. Physical signs, namely, *tenderness* and slight *muscular*

*spasm* are sometimes evident in the right lower quadrant. Final diagnosis usually must be established by roentgenologic study, utilizing the technic of Brown and Sampson.

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*Treatment.*—Patients with the ulcerative form of enterocolitis should remain at *bed rest*. When the involvement is not extensive, improvement of the intestinal lesions will often progress hand in hand with the healing of the pulmonary disease. Decrease in the amount of sputum and the disappearance of bacilli from bronchopulmonary secretions are of considerable importance since the swallowed sputum aggravates existing intestinal lesions. The *diet* must be soft and low in residue. All seeds, skins and strings should be eliminated. This may be accomplished by requiring that vegetables be pureed and that fruits be limited to the strained juices or stewed fruits. Fat meats and whole grain breads are usually interdicted. It must be emphasized that a low residue diet is ordinarily deficient in vitamins. Addition of *brewer's yeast* or one of the concentrates is therefore desirable to maintain an adequate supply of the vitamin B complex. The *cod liver oil* factors are of particular importance and may be supplied in a mixture prepared by floating one to two tablespoonfuls of oil on the surface of 3 ounces of orange or tomato juice, and administered after each meal. If an idiosyncrasy or marked

distaste for cod liver oil exists, one of the other fish liver oils, such as halibut fortified by viosterol, in capsule form is substituted.

*Drugs* must be administered whenever severe diarrhea is present. Bismuth subcarbonate or kaolin suffices to control mild tenesmus and diarrhea. We have always found it necessary to administer an opiate, preferably either the deodorized tincture or camphorated tincture of opium, when cramps and diarrhea are marked enough to exhaust the patient. A course of ten to fourteen daily intravenous injections of calcium gluconate, 10 cc. of the 10 per cent solution, may also aid in the relief of pain and hemorrhage from the bowel.

*Ultraviolet irradiation* conferred by the sun or a lamp is a time-honored remedy of possible merit. The daily exposures should be kept well within the limit of skin tolerance whatever the source; and the rays should be confined to the abdomen, the chest being well covered.

*Artificial pneumoperitoneum* has attained recent popularity in the management of tuberculous enteritis. Lacking extensive personal experience with the method, we may point out that it has not been demonstrated to possess specific curative effects. The advocates of pneumoperitoneum recommend the injection of 300 to 1000 cc. of air into the abdominal cavity at intervals of three to ten days.

*Laparotomy* is not to be advised as a routine procedure. Indeed it is contraindicated in all save those patients who show symptoms and signs of intestinal obstruction, perforation, or acute appendicitis.

**ANORECTAL TUBERCULOSIS.**—Between 5 and 10 per cent of all ischiorectal abscesses and fistulae in ano are caused by tuberculosis. Differentiation from nontuberculous lesions is difficult on the basis of local examination, so that it is necessary to secure a chest roentgenogram on all fistula patients. In the past the dangers of instituting surgical therapy for tuberculous fistula were overemphasized. Actually the relief of discomfort incident to the proper operative treatment ordinarily benefits rather than harms the patient's general and pulmonary condition. Healing after fistulectomy is surprisingly satisfactory as a rule. Operation should be deferred in

those individuals who have acute or recent lesions in the lung, but may be performed with scant risk in the presence of minimal, moderately advanced, or far advanced chronic pulmonary tuberculosis.

### Tuberculosis of the Respiratory Passages

**TUBERCULOSIS OF THE LARYNX.**—Tuberculous laryngitis is a common complication of pulmonary tuberculosis, its incidence being only slightly less than that of ulcerative enterocolitis. Routine observation of the larynx constitutes, therefore, an integral part of the clinical examination of every tuberculous patient.

Not rarely the presenting complaints of the patient are referable to laryngeal involvement. *Impairment of voice* ranging in degree from slight huskiness to complete aphonia is the most common symptom. Deep ulceration and infiltration are capable of producing *pain*, which may be referred to the ear or pharynx, and may also occasion appreciable difficulty or distress on the swallowing of food. Obstructive *dyspnea* seldom ensues in tuberculosis of the larynx, but we have witnessed the development of sudden respiratory difficulties as a result of edema superimposed upon the chronic laryngeal disease.

Tuberculosis shows a predilection for the posterior half of the larynx, the special sites of election being the interarytenoid sulcus, arytenoids and aryepiglottic folds. In addition the true and false cords and epiglottis are often attacked. The gross pathological appearance is that of simple edema or infiltration in early lesions, ulceration and extensive granulation occurring with more advanced disease. Recognition of the tuberculous etiology is based upon the situation and general character of the lesions plus the demonstration of active pulmonary tuberculosis. Biopsy should be performed in doubtful cases.

**Treatment.**—The two most important principles of treatment are: (1) *control of the associated pulmonary disease* by a rest regimen supplemented when necessary by collapse therapy, and (2) *protection of the larynx from trauma* by enforced voice rest. Conversion of the sputum from positive

to negative is usually followed by a striking improvement in the associated laryngeal disease. We may, therefore, regard the management of the pulmonary condition as of primary importance. *Vocal rest* is best attained through requiring written communication. Since whisper is relatively non-traumatizing to the laryngeal structures, permission to hold whispered conversation is sometimes granted. Experience demonstrates that such whispering privileges are often abused, however, so that insistence upon the use of pad and pencil is a wise policy. Smoking ordinarily irritates a damaged larynx and increases any tendency to cough, so that avoidance of tobacco should be strongly advised.

In those individuals who develop extensive ulcerations or large granulomatous masses, *local treatment* to the larynx may be indicated. It has always seemed dangerous to us to instill irritant substances such as chaulmoogra oil or lactic acid into the larynx. The distribution of these solutions cannot be satisfactorily controlled, and they may reach the alveoli of the lung where they are capable of causing inflammation. The actual cautery in experienced hands is certainly more effective in the treatment of ulcers and the reduction of tuberculomas, and its action is restricted to the points of application in the larynx.

Pain and dysphagia may be so great as to demand *symptomatic treatment*. Frequently the application of the cautery to tuberculous ulcers or masses will alleviate distress. One or two aspirin tablets chewed and allowed to dissolve in the mouth may temporarily diminish painful dysphagia. Severe pain usually necessitates the employment of a local anesthetic spray or the injection of the superior laryngeal nerve with alcohol.

**TRACHEOBRONCHIAL TUBERCULOSIS.**—In the past decade increasing attention has been accorded to the development of bronchial and tracheal disease in patients with pulmonary tuberculosis. These lesions range in extent from small discrete tubercles to large ulcerations or granulomatous masses. The importance of these lesions stems directly from their capacity to cause obstruction of the airway. *Asthma-like wheezing*, the appearance of enormous thin-walled or *balloon cavities* in the

pulmonary parenchyma, *lobar atelectasis*, and the *persistence of a positive sputum* in the absence of x-ray evidence of extensive lung involvement are suggestive of tuberculosis of the bronchi. An accurate diagnosis can be made only by *bronchoscopy*, tissue being removed for microscopic examination when indicated.

Suppuration with the production of bronchiectasis or lung abscess may ensue in the bronchopulmonary segments distal to an obstructed bronchus. Another unfortunate attribute of tracheobronchial tuberculosis is its propensity to interfere with the successful application of lung collapse measures in situations which demand such treatment for extensive parenchymal involvement. *Bronchoscopic treatment* with the use of the actual cautery or caustic solutions may be beneficial.

**TUBERCULOSIS OF EAR, NOSE AND THROAT.**—Two peculiarities, namely, absence of pain and the occurrence of multiple perforations of the tympanic membrane may point toward a tuberculous etiology of *middle ear disease*. The most common symptoms are a sensation of fullness and progressive loss of hearing. Operative management is ordinarily not indicated unless there is concomitant extensive involvement of the mastoid.

*Nasal tuberculosis* is rare. Occasionally cutaneous tuberculosis of the face (*lupus vulgaris*) may extend inward to involve the mucosa and even the cartilaginous portions of the nose. Granulomatous lesions of the *nasopharynx* have also been observed; and it has been pointed out that these processes may by themselves be capable of maintaining a positive sputum.

Painful ulcerations of *tongue*, *lip*, or *pharynx* are sometimes seen. In general the ulcers follow the pattern of tuberculosis *cutis orificialis*, exhibiting deep linear fissures with yellow-white bases. Cauterization is perhaps the most effective treatment.

*Tonsillar tuberculosis* often occurs independently of a positive sputum. The involvement varies from microscopic lesions recognized only on routine examination of removed tonsils to grossly visible caseation and ulceration. Ultraviolet radiation to the tonsils and enlarged lymph nodes is often a helpful treatment.

## II. EXTRAPULMONARY TUBERCULOSIS INITIATED BY BLOOD OR LYMPH STREAM DISSEMINATION OF ORGANISMS

It must be emphasized that tuberculosis is a systemic disease, characterized by a more or less widespread dissemination of tubercle bacilli from the primary foci of invasion. During the initial stage of tuberculous infection, invasion of the regional lymphatic structures occurs with great regularity. At the same time, dispersion of bacilli through blood or lymph channels to distant organs may take place. Although generalization of tuberculosis is most common during the primary phase of infection, it is not confined to this stage and may occur or recur at any period subsequent to the first infection.

Autopsy observations demonstrate that the foci produced by such lymphohematogenous dissemination probably heal without event in the majority of individuals. Not rarely these metastatic tuberculous foci become reactivated or progress from the moment of their inception to produce the phenomenon of tuberculous disease of an organ inaccessible to primary tuberculous infection. Tuberculosis of the kidney, genitals, bones and joints, lymph glands, meninges, and adrenals belong in this category. In other instances a massive invasion of the blood stream may evoke the syndrome of miliary tuberculosis.

### Miliary Tuberculosis

Extensive blood stream invasion by the tubercle bacillus produces a highly fatal illness which has been designated acute miliary tuberculosis. Children are more frequent victims than adults, and the condition is commoner in the colored than in the white race. The *onset* may be insidious with a variable period of malaise and general debility, but an abrupt beginning with chill is equally frequent. Eventually the patient presents evidences of *profound prostration*, *rapid wasting*, and *high remittent fever*. The appearance of the patient coupled with the typical low leukocyte count often lead to confusion with typhoid fever. The *diagnosis* of miliary tuberculosis is usually established by the increasing signs of pulmonary involvement, particularly by the roentgen visualization of innumerable tiny dots dispersed throughout the lung fields.

On occasion the first positive clue is the development of effusion in a serous cavity or the onset of meningitis.

It should not be supposed that miliary tuberculosis is an invariably fatal disease. Departure from the characteristic unfavorable course is most common when the bacteremia is limited chiefly to the lesser or pulmonary circulation.

### Renal Tuberculosis

Tuberculosis of the kidney is initiated by the hematogenous deposition of bacilli in the renal cortex. The cortical lesions show a marked tendency to spontaneous regression, however, and progressive renal tuberculosis is a result of extension of the disease into the medullary pyramids and pelvis. Necropsy statistics indicate that approximately 10 per cent of individuals dying of pulmonary tuberculosis exhibit gross renal lesions. Tuberculosis of the kidney need not be accompanied by active tuberculous disease of the lung; in fact pulmonary involvement is insignificant in about one half the cases encountered in noninstitutional practice.

*Diagnosis.*—The first symptoms are usually those of *frequent and painful urination*. Since these signs depend upon bladder invasion established by inoculation with bacilli borne in the urine, they may not properly be regarded as early symptoms of renal tuberculosis. Occasionally gross *hematuria* occurs as an early manifestation. Urinalysis may reveal the presence of pus, albumin and red blood cells, but the pathognomonic characteristic is *the presence of the tubercle bacillus in the urinary sediment*. The organism should be sought in smears of the sediment obtained from the centrifugation of large quantities of urine. Guinea pig inoculation is advisable either to confirm the nature of the bacillus or to demonstrate the organism when the smear is negative.

*Treatment.*—An appreciation of certain fundamental peculiarities of behavior of renal tuberculosis is essential to the proper management of the disease. First, clinical tuberculosis of the kidney is predominantly a *unilateral* disease in its early stages. Secondly, the chances of spontaneous healing of a grossly infected kidney are scant, whereas the likelihood of invasion of the second kidney is considerable so long as the

diseased fellow is retained. In the third place the bladder is relatively resistant to tuberculosis and lesions there show a striking tendency to recover after extirpation of the infected kidney and ureter. These considerations establish the desirability of *nephrectomy* as a treatment for renal tuberculosis. Complete urologic study is of course necessary to exclude tuberculous involvement or functional incapacity of the opposite kidney. The operation may be performed in the presence of quiescent or improving pulmonary disease, and must be deferred only when the lungs are the seat of progressive tuberculosis.

### Genital Tuberculosis

*Genital Tuberculosis in the Female.*—The *fallopian tubes* are the usual primary site of genital tuberculosis in the female, invasion of endometrium or ovaries being sometimes associated. The findings on pelvic examination are not dissimilar to those of gonorrheal salpingitis, but the symptoms are seldom so prominent. Differentiation depends upon recognition of pulmonary and constitutional symptoms of tuberculosis and upon the exclusion of gonococcal infection. Extirpation of extensively involved tubes is the preferred treatment when the general condition of the patient is satisfactory, because it eliminates diseased tissue and prevents spread of tuberculosis to the peritoneum. It should be emphasized that operation is dangerous when peritoneal extension has already taken place, since injury to the bowel or dissemination of tuberculosis is likely to follow attempts to separate the densely adherent tissues.

*Genital Tuberculosis in the Male.*—Male genital tuberculosis is often associated with infection of the urinary tract, but it may be independently established by the hematogenous route. Clinically the infection is usually first evident in the *epididymis* with the development of a soft or fluctuant swelling. Further evolution of the lesion results either in the formation of a chronic draining scrotal sinus or the production of a hard, irregular, calcified mass. In the course of time involvement of the *prostate*, *seminal vesicles* and *opposite epididymis* will usually be demonstrated. The testes are re-

sistant and ordinarily remain intact. Pathological studies suggest that the prostate and seminal vesicles are involved primarily in the genital system, and that epididymal disease represents an extension from these foci by way of the vas deferens. The prostate and vesicles are accessible to palpation only to a gloved finger inserted in the rectum, and thus may escape the attention of both patient and physician.

The conservative surgical procedure of *epididymectomy* seems of definite value in preventing infection of the opposite epididymis, and it has been our policy to recommend this operation in most cases. Some surgeons report good results following the radical excision of prostate, vas, seminal vesicle and epididymis, a procedure with which we have not had experience. Conservative treatment by *ultraviolet irradiation* of an infected epididymis or prostate undoubtedly has a place in the therapy of genital tuberculosis.

#### Tuberculosis of Bones and Joints

Tuberculous osteitis has been considered preeminently a disease of childhood. Bone and joint tuberculosis is by no means a rarity in the adult however, although the pathogenesis apparently differs in the older age group. In children the disease is frequently milk-borne, the osseous lesions representing a bacteremic spread from a primary focus in the gastrointestinal tract. The lungs are usually free of progressive disease in the osteitis of childhood; in contrast, adults who develop Pott's disease or tuberculosis of other bones commonly manifest obvious evidences of pulmonary involvement. Tuberculosis like staphylococcus osteomyelitis commonly begins in the metaphysis of long bones. Unlike pyogenic infections, however, tuberculous osteitis exhibits a marked tendency to invade the neighboring epiphysis and adjacent joint. The tubercle bacillus further displays a marked predilection for elective localization in the vertebrae which constitute an uncommon site of pyogenic osteomyelitis. The most frequent regions affected by osseous and articular tuberculosis are: the dorsal and lumbar portions of the vertebral column, the hip, knee and ankle.

*Diagnosis.*—The diagnosis of tuberculous osteitis or arthritis

is suggested when the clinical manifestations of osseous disease are *insidious and gradual* in their onset. Severe pain is not an early symptom, and at this stage the examiner may detect merely a stiffness or resistance to movement of involved joints. or in the case of freely accessible joints, such as the knee and ankle, demonstrable swelling may be present. *Radiographic findings*, which may be indeterminate at onset, later clinch the diagnosis. *Late signs* include the development of cold abscess in the soft tissues or the appearance of draining sinuses. Recognition of Pott's disease is facilitated by a heightened clinical suspicion based upon the knowledge that tuberculosis is by far the most common cause of spinal disease during childhood and young adult life. During the first decade of life the diagnostic Mantoux tuberculin test is of great value; from this age onward the significance of a positive cutaneous reaction progressively declines.

*Treatment.*—Prolonged *immobilization* of affected joints plus a general *hygienic regimen* comprise the fundamentals of therapy. In children with *spinal tuberculosis*, prolonged fixation may be maintained by the use of a long, bivalved plaster cast, the patient lying on the posterior shell of the cast and being turned at intervals to the prone position with the anterior half of the cast holding the spine in alignment. It is necessary to construct casts or supports in such fashion as to maintain a position of slight hyperextension. Immobilization must often be maintained for years and protection should be conferred by a plaster jacket or back brace after weight-bearing is resumed. In the adult, operative spinal fusion is ordinarily the most satisfactory treatment. It saves time and economic loss and gives more successful end-results on the average.

Immobilization also constitutes the keystone of therapy for other tuberculous bone and joint involvements. Again the most successful end-results are secured when ankylosis occurs, which necessarily means loss of normal joint function. *Heliotherapy* or *ultraviolet irradiation* have been extensively employed in Europe, and it seems certain that such measures are valuable for the treatment of draining sinuses. The management of cold abscesses is discussed on page 612.

## Tuberculosis of Lymph Glands

Tuberculous involvement of the regional lymphatics is a regular concomitant of the primary tuberculous infection. Fortunately the lesions are usually self-limited in extent, and specific therapy other than maintenance of rest during the febrile stage of the primary complex is unnecessary. Occasionally tuberculous tracheobronchial glands may harbor viable organisms for years, and under these circumstances they retain a volcano-like potentiality for producing disaster. For example, rupture of such a gland into a bronchus or through its capsule into the mediastinum may cause a tuberculous pneumonia or a mediastinal infection.

*Generalized tuberculous adenitis* signifies a hematogenous dissemination of bacilli. We have seen this condition principally in colored patients. It should be differentiated from the relatively benign condition of sarcoidosis and from the lymphoblastomas. Excision of a node for examination is usually required except in those instances in which draining sinuses are encountered. The prognosis of generalized adenitis is unfavorable, not because of the glandular lesions, but on account of the miliary dissemination of disease which usually accompanies them.

*Cervical lymphadenitis* presents the most frequent clinical problem. This form of tuberculosis is often produced by the bovine tubercle bacillus, and it has shown a notable decrease in incidence with better control of milk supplies through examination of cattle and pasteurization of milk. The majority of patients escape pulmonary tuberculosis, and the constitutional signs and symptoms associated with cervical adenitis are characteristically mild. *Rest treatment* should be prescribed during the early phase of glandular tuberculosis, and should be maintained until fever or other constitutional symptoms have disappeared and until the progress of the lymph node disease appears to be checked.

Daily exposures to *ultraviolet light* are helpful, but should be avoided during febrile exacerbations. When subsidence of cervical glandular enlargement does not ensue under rest and ultraviolet, *roentgen therapy* may be tried. The x-rays may aggravate caseation necrosis, their most salutary action being

attained in those cases where the nodes are moderately firm to palpation. *Surgical extirpation* of involved glands, once practiced extensively, is necessary in a minority of patients who do not respond to the more conservative procedures enumerated.

### Tuberculosis of the Meninges

Tuberculous meningitis is predominantly a disease of children. Frequently meningeal involvement is a part of the picture of miliary tuberculosis, still more often it begins insidiously in young people who have previously appeared well, and occasionally meningitis develops in adults with chronic pulmonary phthisis.

The *diagnosis* is established by the spinal fluid findings, which include: (1) increase in cell count, lymphocytes predominating, (2) decrease in spinal fluid chloride and glucose concentrations, and (3) the frequent phenomenon of coagulum or pellicle formation when the fluid is permitted to stand in a tube. Pyogenic meningitis is readily distinguished by the great number of polymorphonuclear cells in the spinal fluid. It is much more difficult to differentiate tuberculous meningitis from meningovascular syphilis, encephalitis and acute, benign lymphocytic choriomeningitis. The important differential points comprise: (1) the spinal fluid serology in syphilis, (2) the normal spinal fluid chloride and glucose concentrations in encephalitis, and (3) the prompt recovery of the patient with lymphocytic choriomeningitis.

Meningeal tuberculosis is almost invariably fatal, no effective treatment having been discovered.

### Adrenal Tuberculosis

Tuberculosis of the adrenals is the most common cause of *Addison's disease*. Adrenal insufficiency is suggested by the triad of *asthenia*, *pigmentation* and *hypotension*; and, in doubtful cases, the diagnosis may be based upon the effects of a test period of salt deprivation. The patient with Addison's disease is unable to conserve salt, and the normal decrease in urinary chloride excretion does not follow institution of the test regimen. Prolonged restriction of sodium chloride intake

also results in an exacerbation of the symptoms and may precipitate an adrenal crisis. Adrenal tuberculosis may often be distinguished from Addison's disease caused by simple atrophy of the glands by the demonstration of pulmonary tuberculosis or by the roentgen visualization of adrenal calcification in the former.

The associated pulmonary lesions have usually been quiescent in our cases. Adequate *rest* is indicated, nevertheless; in fact it is regularly demanded by the limitation of physical endurance conferred by the disease. When hypo-adrenalism is mild in degree, satisfactory control of symptoms may be achieved by raising the salt intake and diminishing the potassium content of the *diet*. Usually the parenteral administration of either a potent preparation of the *cortical hormone* or the synthetic compound, *desoxycorticosterone acetate*, will be necessary.

### III. EXTRAPULMONARY LESIONS ESTABLISHED BY EXTENSION TO CONTIGUOUS STRUCTURES

#### Serous Membrane Tuberculosis

Although tuberculosis of the serous membranes is commonly the consequence of spread of infection from contiguous structures, it must be granted that the serosae are also invaded by way of the blood and lymph streams in certain cases. Occasionally a simultaneous involvement of several serous cavities (*polyserositis*) may be encountered, a circumstance which appears indicative of a hematogenous pathogenesis. Experimental studies have further shown that effusions may be produced in sensitized animals by the injection of bacillus-free tuberculin into one of the serous cavities, suggesting the possibility that allergy may play some part in the genesis of effusions.

**TUBERCULOSIS OF THE PLEURA.**—Fibrinous (dry) and serofibrinous (wet) pleurisy are usually caused by tuberculosis provided that those cases accompanying pneumonia are excluded. It must be remembered, however, that tularemia, undulant fever, rheumatic fever, pulmonary infarction, malignancy and pyogenic infections of the lung may all produce

pleural effusion. A workable general policy consists in attempting to exclude these conditions by appropriate studies, regarding every pleurisy in which this can be done as tuberculous in etiology. Guinea pig injection of the centrifuged fluid sediment furnishes additional proof when the results are positive. The animal inoculation test possesses two distinct disadvantages: first the time required to ascertain the results, and secondly the fact that it some times fails to demonstrate the organism in proved cases of tuberculous effusion.

*Pain* aggravated by respiratory acts is the most common symptom of pleurisy. The more severe pleural inflammations evoke a fluid exudate as a rule; and, if this is sufficiently great in volume, there may be appreciable *dyspnea*. It is not generally appreciated that tuberculous pleural effusions may begin abruptly with *chill*, succeeded by a *high continuous fever*, which may reach 104° F. or above. A misdiagnosis of pneumonia is often made in these acute cases, an error which can be avoided by supplementing the clinical study with roentgen films.

*Treatment.*—Dry pleurisy should be treated by adhesive strapping to relieve pain plus rest in bed for a month. When the lungs are obviously diseased, the treatment becomes that of the underlying pulmonary condition.

Varying practices are observed by different physicians in the management of *pleural effusions*. There exists controversy as to whether to tap the fluid and as to the desirability of converting every effusion into a pneumothorax. The procedure to be described has been satisfactory in our hands as well as those of other physicians. Thoracentesis is performed after a considerable quantity of fluid has accumulated, the operator emptying the pleural space as completely as possible if the patient's condition permits, and injecting a small amount of air, not to exceed 250 cc., during the withdrawal. A roentgenogram of the chest is secured immediately following the tap. If there is a definite lesion in the homolateral lung, artificial pneumothorax treatment is maintained, fluid being removed and air injected at appropriate intervals. If the lungs are clear, the patient is kept at rest for a period of six months and then gradually rehabilitated. If a considerable quantity of

fluid reaccumulates, we perform thoracentesis a second time regardless of whether or not symptoms demand the tap for respiratory relief. The rationale of this second thoracentesis consists in the proved fact that partial organization of the exudate may otherwise occur, resulting in contraction of the ipsilateral side of the chest and a reduction of vital capacity. All pleurisy patients should receive periodic reexaminations of the chest.

Purulent pleural effusions or *empyemas* may also be produced by the tubercle bacillus. The usual mechanism of pathogenesis consists in the rupture of a caseous nodule near the surface of the lung. The majority of tuberculous empyemas occur either as a complication of spontaneous pneumothorax or artificial pneumothorax in the tuberculous patient. Therapy must take into account both the necessity of collapsing the diseased underlying lung and the obliteration of the empyema space. In some instances this can be accomplished by repeated aspiration and irrigation of the pleural space. The majority of affected individuals must undergo thoracoplasty to achieve cure. Drainage of tuberculous empyema is never practiced unless there is a mixed infection with pyogenic organisms demonstrable by culture of the fluid.

*Spontaneous pneumothorax* may complicate pulmonary tuberculosis, but tuberculosis is not the most common cause of spontaneous pneumothorax. In the majority of cases this accident occurs in healthy young men, who recover promptly and do not manifest subsequent evidence of pulmonary disease. In the author's series of forty-six cases of spontaneous pneumothorax, twenty-eight were of this benign variety. Only six instances of spontaneous pneumothorax in tuberculous patients were observed, and in each of these cases a tuberculous effusion or empyema developed rapidly. The management is that of tuberculous empyema.

**TUBERCULOSIS OF THE PERITONEUM.**—Two varieties of peritoneal tuberculosis, the *ascitic* and *plastic*, are generally recognized. They are not necessarily sharply differentiated, since the plastic or adhesive form may be a sequel to the more severe infections of ascitic or exudative type. Differential diagnosis includes distinction from all other causes of ascites,

carcinomatosis of the peritoneum and hepatic cirrhosis being the most frequently confused conditions. The prognosis depends to a large extent upon associated lesions. In the female, disease of the fallopian tubes is often present and constitutes the focus from which the peritoneum is infected. Intestinal tuberculosis, involved mesenteric glands, or tuberculous seminal vesicles are other possible primary sites of origin.

*Evacuation of fluid* is indicated for diagnosis and on occasion for relief of distention. *Rest therapy* should be maintained for a considerable period of time, preferably longer than in the management of pleural effusions. The other usual *general measures* such as a nutritious diet and vitamin supplements, aided by ultraviolet irradiation in the afebrile stages are also employed. For many years surgeons have noted the apparent beneficial effects of *laparotomy*, but there does not appear to be reason sufficient for recommending this procedure when the diagnosis has been proved. If the operation is undertaken, care should be exercised to avoid separation of adhesions because of the danger of injury to the bowel.

### Cold Abscesses

The tuberculous cold abscess comprises a collection of pus in the soft tissues produced by the spread of infection from an involved bone, joint, or gland. In actuality the abscesses are not cold, and one may often detect an elevation of the temperature of the overlying skin. The characteristic redness and striking warmth of pyogenic infections are, however, lacking. The most common sites of cold abscess are the fascia overlying the psoas muscle and the anterior surface of the chest wall. From the former position the infection, which has usually originated in a carious vertebra, may migrate downward to the fossa ovalis or lateralward into the tissues of the abdominal wall. Cold abscess of the chest wall may stem from an infected rib, cartilage, or caseous parasternal gland.

*Secondary infection* of a cold abscess, detectable by a growth of pyogenic organisms on ordinary culture media, is a serious complication. Open drainage of the purulent accumulation must, therefore, be avoided. It is proper to *aspirate* these abscesses through a needle introduced obliquely through

the skin at the side of the lesion rather than over the summit of the abscess. Aspiration is of course very difficult in the case of the psoas abscess and is impossible in the average paravertebral abscess. A most important part of the management consists in the treatment of involved bones and joints by immobilization.



## X-RAY ASPECTS OF TUBERCULOSIS OF IMPORTANCE TO THE GENERAL PRACTITIONER\*

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THE lungs lend themselves very readily to roentgen examination. Air filling the bronchial tree and alveolar structures provides the necessary contrasting medium for a clear delineation of the solid lung structures. Small areas of infiltration involving the lung structures will become visible in the roentgenogram because of the surrounding air content of the lung. The ability to visualize a lesion depends upon its size and location; microscopic lesions of course cannot be visualized, but it may be safely assumed that, in most cases, lesions in the parenchyma large enough to be visualized at autopsy should be large enough to be seen in the roentgenogram.

There can be no doubt today as to the value of roentgenographic examination of the chest in pulmonary tuberculosis. It is universally accepted as the most valuable single method of examination. *Stereoscopic* examination is of special value in detection of very slight lesions since it aids in differentiating structures overshadowed by bones; for well established cases however, the stereoscopic method is by no means essential for diagnosis or observation of progress of the disease. In general it may be said that roentgen examination usually discloses more extensive disease than is expected from other methods of examination. In miliary pulmonary tuberculosis it is the only method by which the diagnosis can be made, since the tiny widespread lesions produce no distinctive clinical signs.

*Serial* roentgenographic examinations made at frequent intervals during the disease afford a record of the progress of the disease which can be studied over long periods during

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the patient's illness, providing information as to healing of the lesions by fibrosis, closure of cavities, absorption of exudate and development of complications such as pleural effusion and pneumothorax.

The two methods of roentgenologic examination at our disposal are roentgenography and fluoroscopy. Both have advantages and disadvantages. The *roentgenographic film* is of course the essential method in all cases since it presents in greater detail the finer lung structures. In addition it has a decided advantage in that it leaves a permanent record for future study or comparison with subsequent roentgenograms, a condition which is essential in following the progress of a pulmonary tuberculous lesion.

*Fluoroscopy*, on the other hand, cannot be relied upon to produce the fineness of detail often necessary for the diagnosis of minimal tuberculous lesions. It offers only a transitory view of the lung fields and does not permit proper time for study or evaluation of the shadows produced; it leaves no permanent record for comparison. Its only advantage is that it offers a medium for observation of the function of moving parts such as the chest wall and diaphragm during respiration, and the action of the heart. Best results are obtained by judicious use of both methods whenever they are indicated.

#### FIRST INFECTION WITH PULMONARY TUBERCULOSIS IN CHILDREN

Tuberculosis being so prevalent, first infection most frequently occurs in early childhood, although of course it can occur at any age. The roentgen findings merely reflect the underlying disease process in the lung. In this condition the infective agent, breathed in through the air passages, lodges in the terminal alveolar structures and begins to grow. There does not seem to be any site of predilection; the initial lesion may just as likely fall in the upper as in the lower portion of the lung. Lymphocytes and body tissue cells attack the invader attempting to rid the body of the infection, and a tiny tubercle is formed. Such small lesions may be seen roentgenographically, but may not be recognized as of pathologic significance. The tubercles are usually single but may be mul-

tuple, and they may occur at any location in the lung. They may be so small at this stage that they go unnoticed in the roentgenogram, as is often the case with the initial lesion of first infection with pulmonary tuberculosis in children.

From a practical standpoint, then, we cannot look to the discovery of the initial lesion to provide a reliable diagnostic sign of first infection with tuberculosis in an appreciable percentage of cases (Fig. 106); however, there are other pathologic reactions which may be of aid in the roentgen diagnosis of first infection with tuberculosis. The hilum nodes, draining the area of parenchymal infection, become extremely enlarged and swollen, giving a rounded, nodular appearance to the *hilum shadows* which is readily recognizable in the roentgenogram. If other childhood diseases known to cause tracheobronchial adenopathy, such as measles, whooping cough and Hodgkin's disease, can be ruled out, the diagnosis of childhood tuberculous infection may be safely assumed.

#### Healing of Childhood Lesion

Tuberculous lesions are prone to heal by fibrosis and calcification (Fig. 107): so it is that this small nodule (*Ghon's tubercle*), representing the site of first infection with pulmo-



Fig. 106.

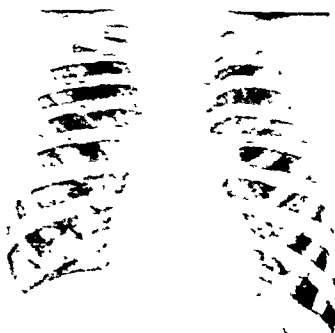


Fig. 107.

Fig. 106.—First infection with pulmonary tuberculosis; as often happens in the active stage the initial parenchymal lesion cannot be identified, but the enlarged hilum lymph nodes give the clue to the diagnosis.

Fig. 107.—Healed lesion of first infection with pulmonary tuberculosis; Ghon's tubercles are readily seen at this stage because of calcification; the hilum nodes become smaller and show areas of calcium deposit.

nary tuberculosis, often goes unrecognized in the roentgenogram until healing with calcification renders it detectable as a pathologic lesion. With healing of the lesions the glands also lose their large, smooth, rounded appearance, becoming contracted and irregular in outline and showing deposits of calcification. That an overwhelming majority of adults show evidence of healed childhood tuberculous infection is evidence both of the widespread occurrence of the disease as well as the high degree of immunity which the human race must bear to the infection.

### Modes of Extension of Childhood Tuberculosis

While a vast majority of children infected with tuberculous lesions recover in the manner indicated, occasionally an instance is encountered in which this form of the disease process extends to other portions of the lung or even to the entire body. Such extensions may be by continuity of tissue or by way of the lymphatics, the bronchial tree or the blood stream.

What are the roentgenologic criteria, then, which herald the unfavorable advancement of childhood tuberculous infection? Most frequently, advancement of childhood tuberculous infection occurs from rupture of the capsules of the hilum nodes; as long as the capsules of the glands remain intact, even though the gland may be broken down and caseous, it protects the surrounding lung structures from infection. If the gland becomes overwhelmed, however, the infection breaks through the capsule and disseminates into the surrounding lung structure (Fig. 108). The first roentgenographic sign to herald the advancement of a first infection type of tuberculous infection therefore is seen in the hilum shadows; the rounded smooth margins of the enlarged hilum nodes of childhood tuberculosis begin to lose their clear-cut outline and to show irregular zones of adjacent infection. Once the capsule is broken through, dissemination of the tuberculous infection may be very rapid, resulting in extensive tuberculous involvement of the lung. As the infection advances, *linear markings* develop, radiating back into the lung field in all directions like a sun-burst, from the broken-down gland. These linear markings are probably due to retrograde

spread of the infection along the engorged lymphatics, the engorgement of the lymph vessels causing inefficiency of the

Fig. 108.

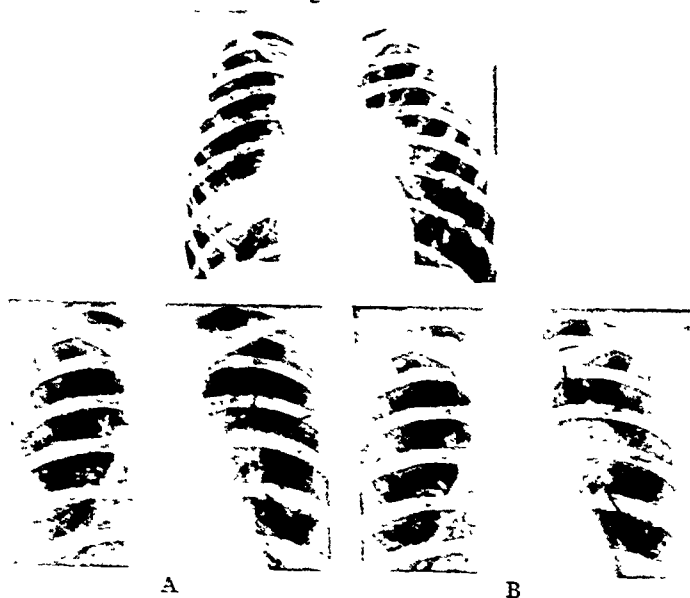


Fig. 109.

Fig. 108.—Breaking down of a caseous hilum gland with spread of the tuberculous process into the lower portion of the right lung field—spread from a lesion of first infection.

Fig. 109.—A, Spread of the tuberculous infection from the hilum nodes into the lung. B, Three months later. Note the extension of the area of infiltration and the cavity which has formed in its center.

valves. The broken-down gland itself may become the site of cavity formation (Fig. 109, A, B).

#### REINFECTION TYPE OF PULMONARY TUBERCULOSIS

Children who recover from their first infection with pulmonary tuberculosis carry scars of their affliction into adult life in the form of calcified nodules in the lungs and calcareous deposits in the regional lymph nodes. Such children may, on being again exposed to tuberculous infection in later life, become reinfected. Reinfection with pulmonary tuberculosis in

adult life takes on a much different picture from that seen in first infection (Fig. 110). The initial site of infection has a predilection for the *apices* or *subapical regions*, first becoming evident as a small cluster of soft, rounded deposits in the parenchyma known as *infiltrations*. Roentgenographically these appear as light, feathery areas in the peripheral lung field in the upper lobes. These infiltrations may at the onset be very small, scarcely more than a few millimeters in diameter, but

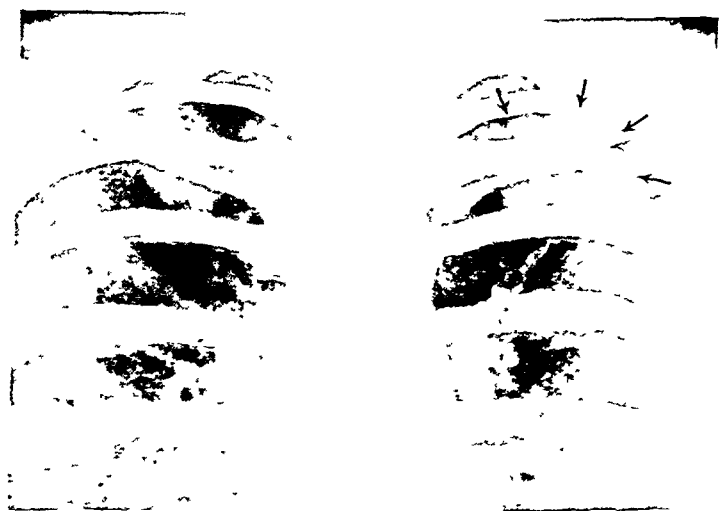


Fig. 110.—Reinfection with pulmonary tuberculosis (in adult life). Note the cluster of soft infiltrative lesions in the left apex and infraclavicular area, and the accentuated lung markings converging fan-shaped toward the hilum; *Minimal Pulmonary Tuberculosis*.

as the disease progresses they enlarge and coalesce, forming *conglomerate tubercles* and small areas of *consolidation*.

The defense mechanisms of the body attempt to rid it of the infection by attacking the invading organisms. Engorgement of the lymphatics draining the infected portion of the lung produces accentuation in the roentgenogram of the lung markings converging fanwise toward the hilum region. Adult reinfection does not have as pronounced an effect on the hilum nodes as that seen with first infection in childhood, consequently the hilum shadow does not present an enlarged

nodular appearance. If an individual escapes infection in childhood, receiving his first infection with tuberculosis in adult life, the characteristics of first infection will be very apt to develop.

Perhaps the explanation for this difference in the mode of infection with tuberculosis as seen in childhood first infection and in the reinfection in adult life may be found in structural differences in the lungs of children and adults or in some difference in their bodily reactions. The structural characteristics of the lung likewise may offer some explanation as to why the tuberculous infection in adults has a special predilection for the parenchymal structures in the apical regions.

#### BODILY REACTION AND SPREAD OF PULMONARY TUBERCULOSIS

Infection with the tubercle bacillus calls forth three types of bodily reaction: (1) *exudation*, (2) *destruction* and (3) *fibrous tissue* formation. These are elements of local reaction, going on in varying degrees at the same time in different portions of the lung. Of these, the first two represent the destructive action of the infection, the last represents a reparative process. Exudation, which is the first response of the body to the infection, produces tiny collections of cells and serum about the invading bacteria and these appear roentgenographically as small areas a few millimeters in diameter with *soft, feathery edges*, located in the periphery of the lung field. These enlarge and coalesce with others to form *areas of consolidation*; this is all a manifestation of exudative reaction (Fig. 111).

As the areas of consolidation become larger, both from toxic reaction and interference with blood supply, their centers undergo necrosis and cavity formation, representing the second type of tissue reaction to this infection (Fig. 112). Roentgenographically this is manifested by the development of a *dark area within the lighter area* caused by the consolidation. With recovery, healing takes place with replacement of the destroyed areas by fibrosis and calcification, thus indicating the third type of bodily response to tuberculous infection (Fig. 113); fibrosis is indicated by the assumption of a

*discrete, clear-cut appearance* by the lesion, and calcification is recognized by its great *density*, which is similar to the density of bone.

By serial roentgenographic examinations taken at intervals during the course of the disease, the extent and character of the lung involvement can be determined. In this manner, any spread of the infectious process can be observed. *Spread of the disease by continuity of tissue* almost always takes place to some extent. The individual lesions become larger and spread to the adjacent portions of the lung (Fig. 111). The



Fig. 111.



Fig. 112.

Fig. 111.—Spread of the tuberculous infection by continuity, forming areas of consolidation, *Moderately Advanced Pulmonary Tuberculosis*.

Fig. 112.—Extension of the tuberculous process to involve large areas of lung structure with dense consolidation in bases of both lungs and cavity formation in the upper portion of the right lung; *Far Advanced Pulmonary Tuberculosis*.

*lymphatics may also serve to spread the disease* by drainage of the infected area both toward the hilum and back over the surface of the pleura (Fig. 114). Lymphatic spread may even be retrograde, since valves in the lymphatic vessels which ordinarily would prevent such backward flow may become incompetent when the lymphatics become engorged. *Spread to some distant part of the lung by way of the respiratory system* (Fig. 117) is one of the most important means of spread of the disease; infectious material, coughed up from one side, gravitates down another adjacent bronchus either on the same side or on the opposite side of the chest and is

aspirated into a new area of the lung. Roentgenographically, the area of new involvement can be seen as a cluster of *feathery, rounded infiltrations* at the periphery of the lung in a region previously free from involvement. Even though the primary lesion which was the source of this infection may be old and appear fibrotic, still the area of recent inoculation takes on the appearance of a soft exudative lesion going progressively through all of the regular stages of the original lesion before final fibrosis and healing occur.

#### ROENTGEN MANIFESTATIONS OF HEALING

*As healing with fibrosis occurs* the individual lesions change from a soft, feathery appearance to one which is more discrete and clear-cut in outline. It is possible, therefore, on examining a roentgenogram to judge by the appearance of the lesions in various parts of the lung as to their relative age and the sequence in which they occurred in the infection; that is, the site of original infection can usually be differentiated from subsequent areas of inoculation by its roentgen characteristics. The relative ages of the different lesions can usually be judged with a fair degree of accuracy from the degree of fibrosis present; however, it may be difficult if not impossible at times to determine complete ultimate healing. While the roentgen manifestations of healing are fibrosis and calcification, it is hazardous from a single roentgenographic examination to attempt to determine the state of activity of a lesion. Although a lesion may appear densely fibrotic, yet some viable areas may still be present in the tissues. From a series of roentgenograms, however, if they show little change over a period of years, it is possible to assume with considerable accuracy that a lesion is at least quiescent. After all, roentgen examination indicates the anatomical extent and character of lung involvement only; the activity of the lesion remains in the realm of the clinician.

#### ROENTGEN MANIFESTATIONS OF DIFFERENT TYPES OF TUBERCULOUS INFECTION

In different types of tuberculous infection, one or another of the three types of reaction may predominate; for instance,

in *acute pneumonic tuberculosis* of both the *lobar* and *lobular* type, the exudative reaction definitely predominates. In certain cases *lung destruction* is very pronounced with *excavation* of large portions of the lung either in association with extensive consolidation or *chronic ulcerative tuberculosis*. In still other types, such as *chronic fibroid tuberculosis*, large amounts of fibrosis occur which limit the advance of the disease.

### Acute Bronchopneumonic Tuberculosis

Pulmonary tuberculosis may be bronchopneumonic or lobular in type, with clusters of *soft, rounded infiltrations* in the parenchyma of the lung occurring in any location, often in the lower portion (Fig. 115). These probably represent lesions of first infection similar to the Ghon's tubercle but more extensive in number and more virulent. The spread of the disease throughout the parenchyma by continuity of tissue is rapid, giving little time for enlargement of the tracheo-bronchial nodes to the degree seen in the usual type of Ghon infection.

At this stage the roentgen findings may give rise to the impression that the condition is a simple bronchopneumonia of ordinary bacterial origin. Groups of soft, rounded infiltrations are seen roentgenographically clustering about the lower lobe bronchi, giving much the same appearance as ordinary bronchopneumonia. The clinical picture likewise may not be decisive. Differentiation cannot be made by sputum examination because children swallow their sputum and examination even of the stomach washings may not at this stage disclose tubercle bacilli. However, infections of this sort are virulent and the spread is rapid; the individual areas of infiltration enlarge and coalesce forming large homogeneous areas of consolidation. The spread may be noted from day to day by serial roentgen examinations. These areas undergo caseous degeneration and within a few weeks or months break down with cavity formation. At this stage tubercle bacilli should be readily demonstrable in the sputum obtained, either by expectoration or gastric lavage. The disease spreads rapidly; caseous consolidation and extensive cavity formation may in-

volve a major portion of both lungs. The condition is usually rapidly fatal, being known as *phthisis florida* or *galloping con-*

Fig. 113.



Fig. 114.



Fig. 115.

Fig. 113.—Fibrosis, manifested by the dense, well-defined character of the lesions, indicating healing by fibrosis. Large cavities are present in both upper lobes; their sharply outlined thin walls are evidence of their chronic fibrotic character.

Fig. 114.—Large tuberculous pleural effusion. The pleural cavity may become involved by retrograde lymphatic spread. In this instance the pressure of the fluid is sufficient to cause displacement of the mediastinal structures to the opposite side.

Fig. 115.—*Acute Bronchopneumonic Tuberculosis*. Multiple areas of infiltration coalesce to form areas of consolidation, on the right side; finally breaking down into cavity formation. Note the cluster of peribronchial infiltrations on the left side from extension by way of the respiratory tract.

*sumption*. It is most commonly encountered in children, but may occur at any age.

### Acute Lobar Pneumonic Tuberculosis

Acute lobar pneumonic tuberculosis is essentially an acute pneumonic process associated with all of the clinical signs and symptoms of lobar pneumonia, and roentgenographically the picture may also be similar (Fig. 116). Diffuse homogeneous consolidation confined to one or more lobes, with usually little if any evidence of previous tuberculous involvement, give a roentgen picture similar in every respect to ordinary pneumococcal lobar pneumonia. Not until the time for

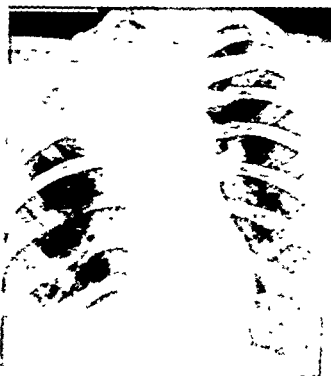


Fig. 116.



Fig. 117.

Fig. 116.—*Acute (Caseous) Lobar Pneumonic Tuberculosis*. Dense, homogeneous consolidation limited to the right upper lobe giving an appearance similar to ordinary lobar pneumonia. After a few months cavity formation occurs and the disease usually spreads throughout the lungs.

Fig. 117.—Complete excavation of entire right lung following pneumonic tuberculosis. Note spread of the infection by way of the respiratory tract across the midline to the opposite side.

crisis is well past, with no change in the patient's condition or in the roentgen appearance of the consolidated area, is the true nature of the disease suspected. Even search for tubercle bacilli may be futile at this stage, since organisms may not appear in the sputum until ulceration and lung destruction occur.

With such large areas of involvement, *cavitation* almost inevitably results in the center of the consolidated area; this usually appears after about two or three months and may ex-

tend greatly. It may in fact proceed to complete excavation of an entire lung (Fig. 117). The right upper lobe is the site of predilection in a large majority of instances, but any lobe or combination of lobes may become involved. The disease occurs most frequently in adults but may occur at any age. When this type of acute pneumonic tuberculosis occurs in children, it is frequently followed by miliary tuberculosis and death; the miliary involvement is not evident at once but comes on from four to six weeks later.

### Acute Miliary Tuberculosis

Acute miliary tuberculosis may develop either in children or adults. In either event its development is the result of inoculation of tubercle bacilli into the blood stream from some previously existing tuberculous focus in the body. In children especially this is frequently due to rupture of a caseous lymph node. The destructive process in the lymph node may erode through a blood vessel, dumping tuberculous material into the blood stream, with the result that the infection is disseminated throughout the portion of the body supplied by the blood vessel. If the lungs should become "seeded" with tubercle bacilli, miliary tubercles would result, uniformly distributed throughout the lung fields; if it happened to be a vessel of the systemic circulation then dissemination might occur throughout the entire body, giving rise to widespread miliary tuberculosis (known as the *typhoid form* because of clinical resemblance to this disease). One should bear in mind the fact that miliary tuberculosis can occur independently in the abdomen, chest or central nervous system, and that it is possible for a person to succumb to miliary tuberculosis involving either the abdominal or central nervous system structures without a miliary tubercle appearing in the lungs. A negative roentgenogram of the chest then does not rule out miliary tuberculosis elsewhere in the body.

Roentgenographically, acute miliary tuberculosis, when it does occur in the lungs, is recognized by the appearance of thousands of *tiny, rounded infiltrations, uniform in size and distributed throughout the lungs*. They should be uniform in size, since all lesions were planted simultaneously, giving all

the same opportunity for growth; they should be uniform in distribution since dissemination by the blood stream provides a method for the even distribution of the infection into the minutest structures of the lung. As time goes on, these tiny infiltrations enlarge uniformly in size, attaining in children a diameter of several millimeters.

Although miliary tuberculosis occurs most frequently in children, it must be considered as a very rare disease; even more unusual is the occurrence of a condition which is most apt to be confused with it roentgenographically, namely *bronchiolitis*. Both conditions show miliary nodules of uniform size, uniformly distributed throughout the lungs; the decisive point in differentiation lies in the fact that with miliary tuberculosis the patient is extremely sick and prostrated whereas in bronchiolitis the patient may not appear very sick at all, or may be playing about in a normal manner. The miliary roentgenographic appearance in bronchiolitis is caused by thousands of tiny fibrous nodules which occur throughout the lungs in the regions of the terminal bronchioles, probably resulting from other types of bacterial infection; at any rate, the degree of toxicity is by no means as great as with miliary tuberculosis and patients usually go on to complete recovery with the formation of minute fibrous nodules which persist for long periods of time.

Once the diagnosis of acute miliary tuberculosis is established in a child, the *prognosis* must be considered as practically hopeless. While there is some evidence to indicate that miliary tuberculosis in the adult may take on a chronic or even a healed form, it has by no means been established that this is true. Individuals are frequently encountered in whom roentgenographic examination discloses numerous tiny calcareous deposits rather uniformly distributed throughout the lungs (Fig. 118). Such cases have been encountered even in very young children. From this it has been assumed that they must have been due to miliary tuberculous infection and that they must represent the healed stage of miliary tuberculosis, but inquiry into the clinical history rarely discloses any period of serious illness of the severity seen in miliary tuberculosis. It may be that these represent healed cases of widespread

initial involvement at the time of first infection with pulmonary tuberculosis in childhood.

### Chronic Ulcerative Tuberculosis

Tuberculosis is essentially a chronic long-drawn-out disease. As ordinarily encountered, the disease progresses by small areas of successive involvement throughout the lungs, each area going through the successive steps of exudation, destruction and fibrosis. During the progress of the disease, *cavi-*

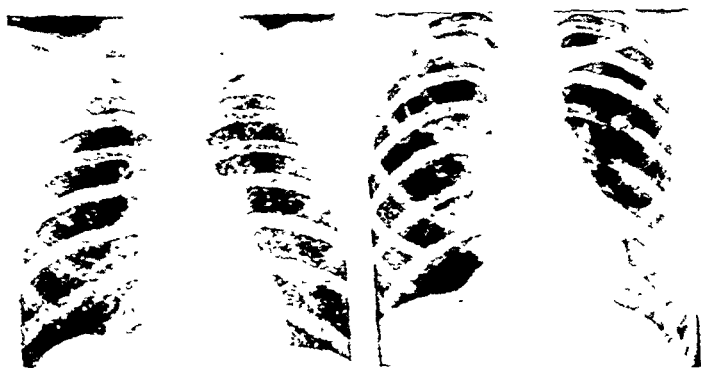


Fig. 118.

Fig. 119.

Fig. 118.—So-called "healed" form of miliary tuberculosis, represented by numerous tiny calcified nodules, uniformly distributed throughout both lungs. While the exact cause is not known, it probably represents a blood stream infection.

Fig. 119.—Large, round, smooth cavity of the chronic "tennis ball" type, so-called because it is thin-walled, rounded and smooth owing to valvelike action of the communicating bronchus which permits air to enter on inspiration but prevents egress on expiration.

*ties* may form which may attain considerable size. This type of tuberculous infection is often referred to as "chronic ulcerative tuberculosis." Roentgenographically, cavities frequently take on a smooth, rounded appearance, with a thin, fibrous tissue wall, and show little change over a long period of time. These are often spoken of as *tennis ball cavities* (Fig. 119). The distended, well rounded appearance of one of these cavities is due to an increase in air pressure within it which results when partial obstruction to its outlet forms a check

valve which allows air to enter the cavity on inspiration and prevents its exit on expiration, thus building up an increase in intracavity pressure. It is obvious that when this results, the very condition itself is not conducive to healing of the cavity.

### Chronic Fibroid Tuberculosis

In the chronic fibroid type of tuberculosis the fibrous tissue reaction of the body to the tuberculous infection is most pronounced (Fig. 113). The formation of large quantities of fibrous tissue aids in restraining the advancement of the disease over a long period of time. Roentgenographically, the large amounts of fibrous tissue formation are indicated by *dense, clear-cut areas* surrounding the tuberculous areas of infection throughout the lungs. This does not preclude the possibility of ulceration of the lesions, however, and all other manifestations of reaction may be present.

### Tracheobronchial Tuberculosis

Ulcerative tuberculous lesions occurring in the bronchial tree may give no manifestations in the roentgenogram; there may be no attending parenchymatous lesions and yet tubercle bacilli may be present in the sputum. Such cases are extremely rare; the diagnosis is made in spite of a negative roentgenogram by finding tubercle bacilli in the sputum; bronchoscopic examination may disclose the ulcerative lesion.

## ROENTGEN AIDS IN TREATMENT

Aside from the information obtained as to the progress and extension of the disease process, there are many other ways in which the roentgen examination may afford aid in the treatment of pulmonary tuberculosis. *Pneumothorax* is probably more widely used today than any other therapeutic measure in the treatment of pulmonary tuberculosis, and roentgenology must be considered as an indispensable aid in carrying out this treatment (Figs. 120 to 124). Roentgenographic examination is of aid, first of all, in determining the suitability of the case for this procedure; marked thickening of the pleura, diaphragmatic adhesions or pocketed pleural effusion may be detected rendering the induction of pneumothorax impossible.



Fig. 120.

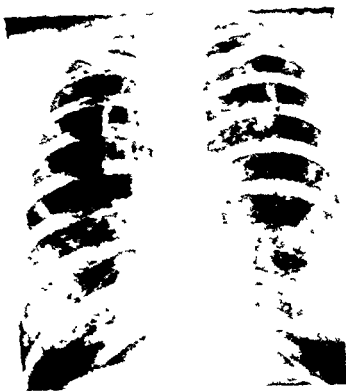


Fig. 121.

Fig. 120.—Pneumothorax with partial collapse of the left lung. Note the areas of consolidation in the partially collapsed lung, and the large cavity uncollapsed even with pneumothorax.

Fig. 121.—Bilateral pneumothorax with partial collapse of both lungs; cavities in upper lobes not fully collapsed show adhesions to parietal wall.

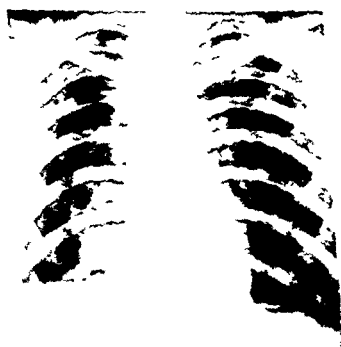


Fig. 122.



Fig. 123.

Fig. 122.—Pneumothorax, right side, with associated pleural effusion. In presence of pneumothorax, fluid takes on a straight line level in the pleural cavity.

Fig. 123.—Pneumothorax with almost complete collapse of lung. Thin strandlike adhesion to parietal chest wall prevents complete retraction of lung. The increased density of the lung is due to associated atelectasis of the lung from bronchial occlusion. The band of adhesions were severed by (cautery) pneumolysis.

During inflation. fluoroscopy provides a visible guide to the degree of lung collapse attained. indicates the presence of any



Fig. 124.

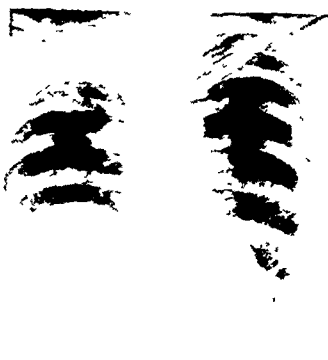


Fig. 125.

Fig. 124.—Extrapleural pneumothorax showing partial separation of the parietal pleura from the chest wall. This was attempted because dense adhesions between the parietal and visceral pleura made ordinary pneumothorax impossible.

Fig. 125.—Phrenicotomy of left phrenic nerve resulting in elevation and immobilization of left diaphragm; induced to try to lessen movement of lung on that side.



Fig. 126.—Thoracoplasty of the left side of the chest with collapse of the chest wall. A large thin-walled cavity still remains on the right side.

restraining adhesions and gives an accurate estimate of the degree of diaphragmatic and pulmonary movement during respiration.

After *phrenicotomy*, fluoroscopic examination shows the height of the diaphragm on the affected side and discloses the degree of its movement during respiration (Fig. 125).

Following operative collapse of the chest wall by *thoracoplasty*, roentgen examination may be of value in showing the degree of success of this procedure in closing cavities (Fig. 126), and later on, after healing takes place, it may disclose the extent of reformation of bone in the resected portions of the ribs.

Special examination of the chest by body section roentgenography may be considered of value in determining the size, distribution and exact location of tuberculous cavities and the detection of enlarged tracheobronchial nodes.

#### ROENTGEN CLASSIFICATION OF TUBERCULOUS LESIONS OF LUNG

This classification, formulated by the American Sanatorium Association and adopted also by the National Tuberculosis Association, is for the guidance of clinicians in their study and treatment of this disease. Since the roentgen diagnosis is one which deals largely with the anatomic distribution and extent of the disease, it must naturally be relied upon to furnish the necessary data for classification in this respect. All cases of tuberculosis under this classification are designated as minimal, moderately advanced or far advanced.

##### *Minimal Pulmonary Tuberculosis* (Fig. 110)

- (a) A lesion showing slight infiltration without demonstrable excavation, or
- (b) A lesion affecting small part of one or both lungs, the total volume of involvement, regardless of distribution, not exceeding the equivalent of volume of lung tissue which lies above second chondrosternal junction and spine of fourth or body of fifth thoracic vertebra.

##### *Moderately Advanced Tuberculosis* (Fig. 111)

- (a) A lesion showing slight disseminated infiltration or fibrosis which may extend through not more than the equivalent of volume of one lung, or
- (b) Severe infiltration with or without fibrosis which may extend through not more than the equivalent of one-third the volume of one lung, or

(c) Any gradation with above limits.

(d) Diameter of cavities if present is not to exceed 4 cm.

*Far Advanced Pulmonary Tuberculosis* (Fig. 112)

A lesion more extensive than under "moderately advanced" or with definite evidence of greater cavity formation.

In addition to these stipulations dealing with distribution and character of the lesions there are others dealing with the clinical manifestations of the disease; these are the realm of the clinician and will be considered by him. The practitioner need not remember the finer details of this classification but should have a working knowledge of it, since it forms the basis of large statistical studies as to prognosis and treatment.

#### THE ROLE OF ROENTGENOLOGY IN PROPHYLAXIS

Strangely enough, the progress which has been made in reducing the death rate from tuberculosis in the past few decades has not been due so much to more effectual methods of treatment as it has to the early detection of the disease and prompt isolation of the patient, thereby preventing the spread of the infection. It would seem, then, that the greatest hope of future progress in reducing the incidence of this disease would be along the lines of prevention rather than cure.

What role does roentgenology play in prophylaxis? It is obvious that, if roentgen examination could be made of all individuals at periodic intervals, the disease could be detected in its earliest stages when it is most amenable to treatment and most readily cured; and, most important of all, the patient could be isolated at once before he has an opportunity to contaminate others. Obviously such a procedure cannot be carried out for the entire population since there are not a sufficient number of x-ray machines or personnel for such an undertaking; and furthermore, the cost would be prohibitive.

#### Miniature Film Roentgen Surveys

In an endeavor to *reduce the cost* of x-ray examination for vast surveys of large schools and other institutions, especially where the individuals concerned are at the ages most suscepti-

ble to tuberculous infection, roentgenology has devised a new method of approach. By proper perfection of speed and quality of x-ray fluoroscopic screens and films; by the construction of more powerful x-ray apparatus and by the development of high speed photographic lens and fine grain photographic film, it has become possible to actually *photograph* the image of the chest on the *fluorescent screen* as it is produced by x-ray exposure.

The great advantage in this procedure is the reduction in cost of the film; it must be recognized at once that such a method, whereby the image on the fluoroscopic screen, which in itself is not as rich in detail as an ordinary 14 by 17 inch roentgenogram when reduced on miniature films, will suffer a definite loss in detail and that they therefore cannot be expected to yield the same degree of diagnostic results. As a *survey method* however, for the *detection of suspicious cases*, it may be invaluable. These patients may then be subjected to conventional roentgenographic examination for the final diagnosis.

Within the next few years the medical practitioner will undoubtedly be confronted with instances in which the school physician, as the result of such miniature film roentgen surveys, advises him that certain children of his clientele appear to be suffering with pulmonary tuberculosis. That *he must not accept such diagnosis on the basis of miniature film surveys as final* is obvious. He should, however, resort at once to the regular 14 by 17 inch conventional film examination such as is used in every roentgenologist's office, since this remains the best, most reliable method for the definite diagnosis of the disease. The advent of the miniature film survey for detecting suspicious cases, however, has made the future loom bright for the ultimate control of this dreaded disease.



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